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DIRECT SUBPERIOSTEAL TRANSCIRCUMFERENTIAL WIRING

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DIRECT subperiosteal transcircumferential wiring is a relatively simple method of obtaining an excellent reduction and a most rigid fixation of certain mandibular fractures.

When mandibular fractures are treated solely by the more routine methods of intermaxillary fixation, it is not uncommon for the surgeon to view the postoperative roentgenograms with mixed feelings of surprise and disappointment. Occasionally he finds that, although he has carefully brought the teeth into occlusion by intermaxillary traction, there is an outward rotation of the inferior border of the mandible. This is especially true if arch bars and intermaxillary elastics have been used, since the force is placed so far lateral to the pivotal centers of the teeth. The more the intermaxillary force is increased, the greater is the displacement.

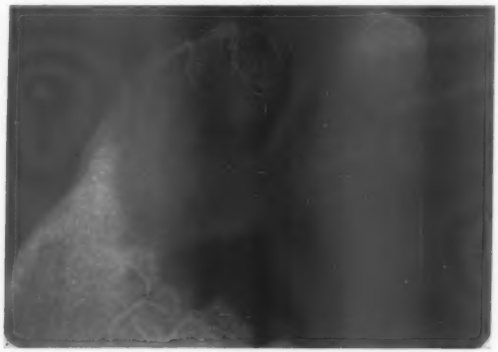
In most cases the discrepancy in alignment is minimal and of little consequence. In other cases, the outward rotation of the inferior border of the mandible is great enough to prolong the healing time and, at best, the buccolingual occlusal relationship of the maxillary and mandibular teeth in the area of the rotated segment will not be ideal. The typical V-shaped opening formed at the inferior border by this type of displacement is shown in Fig. 1.

In attempting to treat fractures by simple wiring or by the use of arch bars, we find that the success of such treatment is dependent upon, among other things, the number of teeth present, their position in the arches, and their firmness or lack of mobility.

INDICATIONS

Direct subperiosteal transcircumferential wiring can be done only in cases of diagonal or oblique fractures in which a wire passing from the superior to the inferior border of the mandible will actually bind the two fractured segments together (Figs. 2 and 3). The procedure is indicated when this type of

Fig. 1.—Roentgenogram showing the typical V-shaped opening that sometimes occurs at the inferior border of the mandible when the fracture is treated solely by intermaxillary traction.



A.

B.

Fig. 2.—*A*, Preoperative topographic occlusal roentgenogram showing that the molar tooth must be removed, leaving an edentulous posterior fragment. Direct subperiosteal transcircumferential wiring is indicated.

B, Postoperative roentgenogram showing direct transcircumferential wire in place. The fracture in the lingual cortical plate gives the illusion of a second complete fracture (refer again to Fig. 2, *A*). A most rigid fixation was obtained in this case.

fracture exists and there are not enough teeth for adequate fixation and immobilization (Fig. 4), as well as when intermaxillary traction causes an outward rotation of the inferior border of the mandible.

This technique does not involve the use of a splint, and it can be employed in either edentulous areas or areas in which teeth are present. This relatively simple method of fixation does not necessarily require hospitalization.

Fig. 3.—Postoperative roentgenogram showing direct transcircumferential wires being utilized in an area in which teeth are present. This patient had very poor teeth, an open-bite in the incisor area, and insufficient maxillary posterior teeth. These factors created a problem which was solved by direct transcircumferential wiring.

DESCRIPTION OF OPERATIVE PROCEDURE

The skin of the face and neck is prepared in the conventional manner. An attempt is made to cleanse the mouth and reduce the bacterial count by applying an antiseptic, such as tincture of Metaphen, to the teeth and oral tissues. Either general or local anesthesia may be used.

If the fracture is in an edentulous area, the incision is made along the crest of the ridge in the area of the fracture. Another incision, perpendicular to the first, is made from lingual to buccal so that the mucoperiosteal flaps can be detached from the bone on both sides of the alveolar process. Maximum exposure on the buccal side is helpful in obtaining an accurate reduction of the fracture under direct vision. The exposure on the lingual side should be minimal.

A 25 gauge wire is introduced by the conventional method with a trocar and cannula which are slightly bent for transcircumferential wiring. The cannula, with trocar in place, is forced through the skin beneath the inferior border of the mandible at a previously selected spot. The cannula is carried

up the lingual surface of the mandible and kept in close contact with the surface of the bone at all times. A blade retractor, such as a Henahan, is used to hold back the lingual flap and guide the cannula.

A.

B.

C.

Fig. 4—*A*, Occlusal roentgenogram showing a displaced fracture of an edentulous mandible.

B, Postoperative result of the fracture shown in *A*. Two transosseous wires across the socket of a recently extracted tooth help the direct transcircumferential wire to maintain a rigid fixation.

C, Bony union in the case shown in *A* and *B*.

When the top of the cannula approaches the occlusal level of the teeth or the crest of the alveolar process, whichever the case may be, the trocar is removed and a 25 gauge sterilized stainless steel wire is unrolled from its spool

and inserted into the open end of the cannula which projects into the patient's mouth (Fig. 5). When several inches of wire appear at the other end, the cannula is withdrawn, leaving the wire in place.

The cannula must be used again to carry the wire up around the buccal or labial surface of the mandible. In anterior fractures where there is sufficient access, the cannula and trocar may be introduced from the intraoral approach and brought down beneath the buccal mucoperiosteal flap to emerge on the skin surface through the same opening as the wire. The trocar is withdrawn from the cannula, and the wire is inserted into the open end projecting through the skin. When the wire emerges through the hub into the mouth, the cannula is withdrawn, leaving the wire in place.

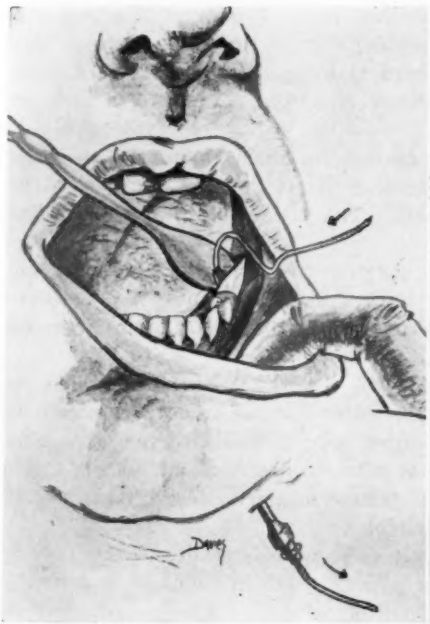


Fig. 5.

Fig. 6.

Fig. 5.—Cannula (or large-gauge hypodermic needle) being used to carry the wire from the mouth down the lingual surface of the mandible to emerge at the opening in the hub.
Fig. 6—Lingual wire drawn through a hole drilled near the crest of the alveolar process to prevent the wire from slipping. Wires are ready to be pulled tight and twisted as the fracture is held in a reduced position.

When there is insufficient access for use of the intraoral approach, the cannula and trocar are introduced through the original opening in the skin and carried up the buccal surface of the mandible to emerge beneath the intraoral mucoperiosteal flap. The trocar is withdrawn and the hub is cut off the cannula before the wire is inserted. This allows the cannula to be removed intraorally. A suitable instrument should be available for reopening the lumen of the cannula after it has been cut with the wire cutter; otherwise, it may be

difficult or impossible to insert the wire. An improved instrument for guiding the wire would be a cannula with a threaded hub that could be removed and replaced as needed.

If teeth are present in the area of the fracture, the lingual end of the wire is passed through an interdental space before the two ends are drawn tightly around the bone and twisted on the buccal side. In edentulous areas the lingual wire is simply passed over the crest of the alveolar process unless there is danger of its slipping into the fracture. If this possibility exists, the surgeon drills a hole through the process near the superior border and inserts the wire through the hole before drawing it tightly around the bone and twisting the two ends together (Fig. 6).

In any case, the wires are twisted so that the knot will lie against the bone at a point beneath the level of the mucobuccal fold. This precaution will prevent the twisted wire from becoming exposed through breakdown of the thin overlying tissues. After the wire is twisted and cut, the knot is pressed down against the bone to help prevent soft-tissue irritation. The flap is then returned to place and sutured.

The wire which has been pulled up against the inferior border of the mandible will cause dimpling of the skin surface unless the tissues are freed by dissection through the opening in the skin. The wound is protected by a film of flexible collodion.

The fracture must be considered a contaminated compound fracture, and the patient should be given therapeutic doses of one of the antibiotics until the danger of infection has passed. Neglecting to administer antibiotics from the first day invites infection and nonunion.

The edentulous mandible shown in Fig. 4, *A* was not immobilized, and yet healing was uneventful (Fig. 4, *C*). The patient whose fracture is shown in Fig. 2 removed all his intermaxillary wires approximately three weeks after they had been inserted. After a week of what he described as "eating steaks like mad," he returned to the office 10 pounds heavier and with his jaw in excellent condition. Healing was uneventful.

These two cases are mentioned merely to show the strength of this type of fixation. Immobilization for four weeks, with the teeth in occlusion, is certainly to be recommended. The transcircumferential wire may be removed after an additional two weeks, or it may be allowed to remain for an indefinite length of time.

Removal of the transcircumferential wire is a very simple matter. Under local anesthesia an incision is made in the mucosa above the area of the twisted wire. A flap is elevated to expose the knot, which is grasped with a hemostat or needle holder. The wire is then cut on one side of the knot and easily withdrawn with a minimum of discomfort to the patient.

CONCLUSION

This technique is presented not as a substitute for the conventional methods of treating fractures by intermaxillary wiring but as an adjunct to be used with intermaxillary wiring where extra help is needed.

If the fracture is of the oblique or diagonal type, in which a wire passing around the superior and inferior borders of the mandible can actually bind the fractured segments together, then direct subperiosteal transcircumferential wiring can be used if indicated by the inadequacy of routine methods of intermaxillary fixation.

The technique is useful in cases in which adequate fixation and immobilization are difficult to obtain because of mobility of teeth or an insufficient number of teeth. The stability of the reduced fracture is far greater than that obtained with transosseous wiring at the inferior border of the mandible.

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SKELETAL TRACTION FOR MAXILLARY FRACTURE

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APPROXIMATION or extension of jaw fractures by means of cords and weights is rarely used. Occasionally a retroposed middle third of the face may necessitate the application of traction by weight from a Balkan beam. A recent case of skull and upper jaw fracture, however, demanded the application of a force distant from the body in order to *approximate* the bony middle third of face to the base of the skull.

CASE REPORT

A 33-year-old man was in a motorcycle accident and sustained extensive fractures of the skull and middle third of the face (Figs. 1 and 2). The dura mater and brain were

Fig. 1.

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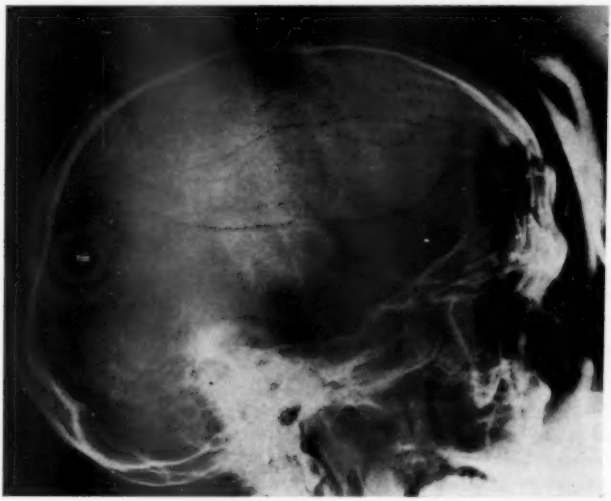

Fig. 2.

Fig. 3.

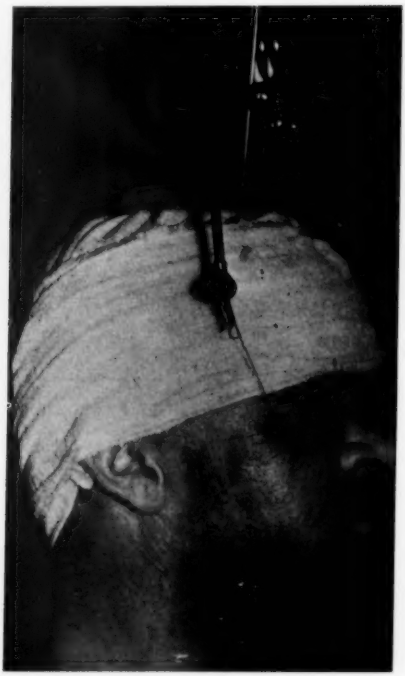

Fig. 4.

extruding. An intracranial operation under local anesthesia was performed by Mr. E. S. Watkins. The upper jaw was so loose and fragmented that it moved to and fro on respiration. Cerebrospinal rhinorrhea was evident. The patient was comatose and noncooperative. Early immobilization of the fragments was necessary in order to control the rhinorrhea, but subjecting the patient to a general anesthetic in order to obtain dental impressions and to fabricate cast-silver splints did not appear justified, especially since there was nearly a full complement of natural teeth which could be used as anchorage.

The considerable edema of the scalp ensuing from the intracranial operation precluded application of a plaster headcap, as the headcap would rapidly have become loose. It was decided to eyelet-wire the teeth, fix the upper and lower jaws in occlusion, and exert traction from the lower-jaw eyelets through cheek wires to a stirrup and cord over a pulley by means of a weight at the head of the bed.

Following induction of endotracheal anesthesia, administered by Dr. G. M. L. Baer, it was found that the palate was fractured near the midline and that the left half was fractured in a coronal plane. Both clinically and radiographically, numerous other fractures of the maxilla and of the malar and nasal bones were seen.

The cheek wires were attached to a femur extension stirrup bent to a suitable shape and carrying a 2 pound weight. In order to prevent the patient from detaching his maxilla from the base of the skull by depressing his mandible, a piece of sheet metal covered with foam rubber was applied to the vertex (which was not fractured) and fixed to the stirrup by locked universal joints (Figs. 3 and 4). The rhinorrhea ceased. Five days later the scalp edema had subsided, and a plaster headcap with conventional attachments was applied.

INCISION AND DRAINAGE: A NEW MODIFIED SURGICAL TECHNIQUE

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THOMA,[6] in his recent book, states that if incision and drainage could be avoided the scar which so often mars the patient's face and remains as a telltale mark of the episode could be prevented in many cases. It has also been recorded that, when incision and drainage is performed, healing is uneventful except for a depressed scar.[1] Such scars may range from adherent, depressed, or hyperplastic scars to dimpling scars (Fig. 1). In order to prevent these scars following incision and drainage of an abscess, I shall discuss the cause of scar formation and, from my clinical study, present a new modified surgical technique.

Fig. 1.—Clinical photograph showing the depressed scar which so often mars the patient's face and remains as a telltale mark of the incision and drainage episode.

CAUSES OF SCARRING

I believe that the scars following incision and drainage are due to (1) use of the conventional technique, (2) packing of the wound, and (3) insertion of rubber catheter tubes for irrigation.

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The accepted conventional technique has been described by Handfield-Jones and Porritt[4] as follows: "The incision should be made if possible at the most dependent part of the abscess to allow satisfactory gravitational drainage."

Thoma[6] states: "The incision should be made where the pus is closest to the surface."

Berman[3] writes: "Should the surgeon elect to open the abscess, he will do so through the center."

Bailey and Love[2] endorse this opinion by saying: "A dependent external incision is indicated."

In the similar way, Archer[1] points out: "It is necessary to wait for 'pointing' of these abscesses before incising for drainage. A soft spot approximately the size of a nickel is frequently found in or near the center of the brawny indurated mass. This is the point to incise for drainage. The spot has the same feeling to the fingertips as a spot in a cantaloupe."

It is a well-known fact that during abscess formation, as the pressure mounts, the pus breaks through the bone, periosteum, muscle, deep fascia, and outer surface of the skin.[5, 6] This causes the skin over the abscess to become tense, shiny, thin, and cherry-red, and it may become necrotic.[3, 5] In view of this, if the incision is made in the "pointing" or at a soft spot in or near the center of the abscess (Fig. 2), naturally the wound will either heal by second intention or result in an undesired scar which leads to considerable deformity (Fig. 3).

Some surgeons believe that the wound should be left open and packed with petrolatum or iodoform gauze so that healing will occur "from the bottom up."[3, 4] Again, in some instances, rubber catheters are inserted to irrigate the abscess cavity periodically or to instill an antibiotic solution.[1, 6] It has been found that these catheters delay healing and incite more scar formation of the adherent type (Fig. 3).

A NEW MODIFIED SURGICAL TECHNIQUE

The object of this technique is to provide an outlet for the pus as soon as possible, so that healing should take place by first intention with minimum undesirable scar formation (Fig. 4).

If the skin over the center of the abscess is thin and tense and shows signs of skin necrosis, the incision should not be made at this point (Fig. 2). Instead, it should be made at the periphery of the abscess, where the skin shows a normal appearance (Fig. 5).

The incision should not be made crosswise to the lines of tension.

The incision must be clean and direct, and it must penetrate the entire thickness of the skin at one stroke and at even depth along its entire length.

The incision should be made at right angles to the surface of the skin in order that the skin edges may meet perpendicularly when healed.

The incision should be ¼ inch long. This is sufficiently large to obtain free drainage.

The superficial and deep fascial layers and muscle fibers should be separated by blunt dissection.

By separating the blades of a curved hemostat, the surgeon should open the various loculi, carefully and gently to avoid injury to the blood vessels and nerves while allowing the entire area to drain (Fig. 5).

Fig. 2.

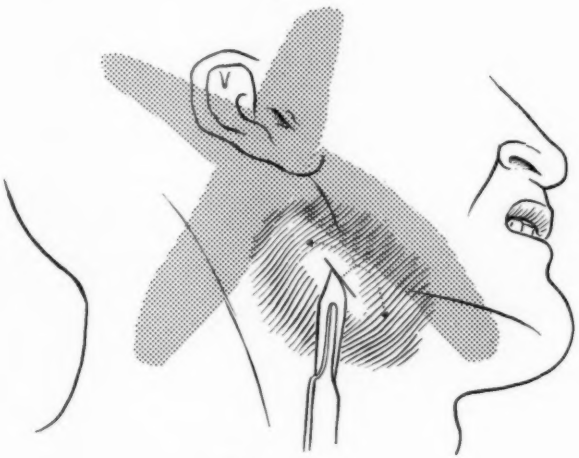

Fig. 3.

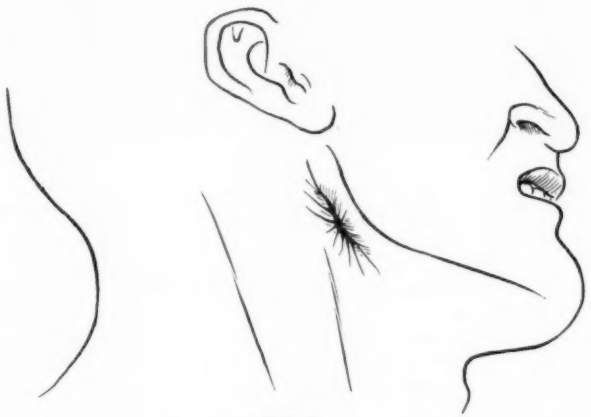

Fig. 2.—Diagram demonstrates the conventional technique showing that incision is usually made at the most dependent part of the abscess.

Fig. 3.—Results of the conventional technique, which leads to puckering of the skin causing considerable tissue deformity.

Careful flushing of the abscess cavity with sterile physiologic saline solution at the conclusion of the operation is recommended.

Having opened the abscess and explored the cavity, one should express all obvious sloughs and a nonabsorbent drain or rubber tissue should be inserted

into the evacuated space to ensure adequate drainage. It is suggested that drains should not be sutured to the skin; instead a knot should be made at one end of the rubber tube which is placed deep into the incision, as this prevents it from slipping out and eliminates suture scarring. The drain must be removed within twenty-four to forty-eight hours following incision and drainage.

Fig. 4.

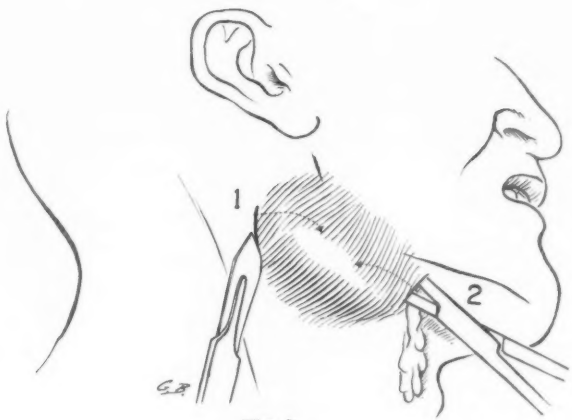

Fig. 5.

Fig. 4.—Results of the new modified surgical technique. Note minimum scarring without tissue deformity.

Fig. 5.—Diagram illustrating a new modified surgical technique. Please note that the skin incision should be made at the periphery (*1* or *2*) of the abscess where the skin shows a normal appearance.

The wound area should be covered with a sterile dressing to prevent contamination from the outside with secondary organisms.

Intermittent application of hot, moist packs over the area and intelligent use of antibiotics are necessary to accelerate wound healing. This is of prime importance. Poultices of all kinds should be avoided.

In addition, one should give attention to the patient's general condition and build up his immunity.

The two case reports that follow are presented from the series of cases which I have treated to show not how to avoid scarring but, rather, how to minimize scarring.

CASE REPORTS

CASE 1.—Patient D. V., a 23-year-old man, came to the oral surgery clinic of the Research and Educational Hospitals in Chicago with a complaint of swelling and pain of ten days' duration in the right jaw (Fig. 6). The case history and complete physical and laboratory examination revealed an acute abscess of dental origin. The swelling was 2 inches in diameter, well circumscribed, fluctuant, and tender, with classic signs of an acute abscess.

Fig. 6.

Fig. 7.

Fig. 6.—Preoperative clinical view of circumscribed mandibular abscess.
Fig. 7.—Postoperative photograph of patient D. V., showing the healing of the wound resulting in an imperceptible scar.

It was decided to incise and drain the abscess and, in the same operation, to extract the right lower first premolar, which presented an apical granuloma and was necrotic. The patient was prepared under a local anesthetic. A skin incision was made at the periphery instead of at the center, as called for in the conventional method. The abscess was drained and treated in the manner described for the new modified technique. The healing of the wound resulted in an imperceptible scar (Fig. 7).

CASE 2.—Patient R. S., a 26-year-old man, was referred to the oral surgery service with a history of recurrent abscesses on the right side of the face. The patient stated that a month earlier he had a recurrent episode of an abscess which was lanced and drained by a surgeon at another civilian hospital in his hometown. He said that his abscess was packed with some sort of gauze and that dressings were changed every other day; the wound healed with apparent scar after two weeks (Fig. 8, white arrow). A fortnight later the swelling recurred; this time the patient received a course of penicillin "shots," but this therapy did not relieve his symptoms. Finally, he was referred to the oral surgery department of the Rajendra Hospital in Patiala for diagnosis and treatment. The patient was seen by the attending oral surgeon, and complete evaluation showed a recurrent abscess of odontogenic

origin involving the right lower carious molar, the x-ray of which showed a periapical radiolucent lesion. The patient had trismus and was in acute pain and agony. His general condition was within normal limits. It was planned that the abscess would be incised and drained first to relieve the acute symptoms and that as soon as the patient could open his mouth the offending tooth would be extracted. The abscess was incised and drained according to the

Fig. 8. Fig. 9.

Fig. 8.—Clinical view of Patient R. S., showing scar resulting from the conventional technique (white arrow) at the first episode. Note that second incision was made according to new modified technique (black arrow).

Fig. 9.—Photograph revealing the difference between the scar formation of the conventional technique (white arrow) and the wound healing by the new modified technique (black arrow).

Fig. 10.—An advantage of the new modified surgical technique is a minimum scar at the site of incision of an abscess.

modified technique already described (Fig. 8, black arrow pointing to drain in place). Four days later the offending molar was extracted under local anesthesia. Note the difference between the scar formation following the conventional technique (Fig. 9, white arrow) and the wound healing following the new modified technique (Fig. 9, black arrow).

SUMMARY

Causes of scar formation following incision and drainage of an abscess have been discussed and a new modified surgical technique which has been illustrated by diagrams and documented by clinical case reports has been recommended. It is suggested that whenever the skin changes are noted at the most dependent part of the abscess, an incision at the center should be avoided (Fig. 2) and drainage should be accomplished by approaching the abscess through the periphery via normal skin (Fig. 5).

Evacuation of the purulent material through the proper surgical approach, with prompt removal of the cause, leads to complete recovery without loss of time and without tissue deformity at the site of incision (Fig. 10).

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REMOVAL OF SUTURES FOLLOWING ORAL SURGERY

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FORMERLY, untreated silk, linen, and cotton threads were used as suture material. During the process of healing, tissue cells grew into and permeated the interstices of the threads. Removal of these materials disrupted newly formed tissue and was therefore a painful procedure. For this reason, many patients learned to dread the removal of sutures.

Nowadays, because of the impervious materials available, removal of sutures should be relatively painless. If there is discomfort, it is probably due to an accident on the part of the operator or to the use of a faulty technique. If the patient remains motionless and the operator is dexterous and follows a standard technique, there is no excuse for the removal of sutures causing pain, damage to the wound, or infection of the suture bed.[2]

The technique of suture removal is simple and can be readily understood after a brief examination of the accompanying illustrations. Ordinarily, only two instruments are needed—(1) a delicate cross-ribbed dissecting forceps and (2) a pair of sharp, fine-pointed scissors. (Some operators use combination suture-cutting scissors, but we prefer ordinary long, tapering scissors with two sharp-pointed ends.)

If the skin wound is small and its edges are in easy and normal apposition, without tension, and if the sutures are doing little mechanical work in holding the edges together, they may be removed on the third or fourth day. On the face, in particular, where scarring is to be minimized, skin sutures can and often should be removed on the third or fourth day. If the lips of the wound are under tension and the sutures are mechanically holding the tissue in contact, they may be left in situ for two or three days more. An alternative is to remove alternate approximating sutures up to the seventh day and then to remove the remaining ones a day or two later. Skin sutures are rarely left beyond the tenth day; in general, sutures closing average septic wounds are usually removed on or about the seventh day. Sutures placed to close average

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uncomplicated intraoral wounds are usually removed between the fourth and the sixth days; alternate sutures may be removed on the fourth day and the remainder on the sixth day.

Before sutures are removed, the suture line should be swabbed with an antiseptic solution (Fig. 1). Then the extraneous part of the suture should be gently grasped with a smooth forceps just beyond the knot (Fig. 2). At right angles to the tissue surface, the suture is lifted directly upward with a rotary motion just far enough to expose the subcutaneous part of the suture (Fig. 3). Now, the sharp-pointed end of the scissors, held flat, is slipped beneath and made to embrace the subcutaneous part of the suture, which is severed transversely (Fig. 4). The suture should not be cut in the middle, as this would necessitate drawing a potentially contaminated piece of suture through the wound. If the wound itself is sterile, the subcutaneous length of the suture is certainly sterile. The final step is to draw the suture *toward* the wound (not vertically upward and certainly not outward, which would actually tend to separate the lips of a weakly united wound) (Fig. 6).

A second technique, although not as much favored, may be more expeditious. The exact middle of the suture is grasped with a forceps, and the two portions of the buried suture are drawn upon equally. A little of the moist concealed part of each subcutaneous suture is brought into view by slight tension on the loop. The tissue on one side of the suture is depressed by means of a pair of sharp-pointed scissors with the blades separated, and the moist buried part is cut. The maintained tension on the free end will bring the opposite end up and draw out the entire length of the buried portion of the suture.

In neither of these techniques is any part of the suture lying outside the tissue drawn backward through the deeper parts. Thus, incrustations and possible infection are avoided.

The withdrawal should be steady and gentle until the entire suture is removed. The scissors may aid in counterpressure during withdrawal by gently pressing the tissue on each side of the remaining extracutaneous portion of the suture (Fig. 5).

After the sutures have been removed, proper support of the wound is essential. Adhesive tape "butterflies" (Fig. 7) should be applied to the incision line for another week to relieve all tension on the suture line.[1] This should be done routinely on the face where it is important that sutures be removed early and that, at the same time, separation of the wound edges be avoided.

Following removal, the suture holes act both as exit drains for any fluid in the wound (which should be rare at this stage) and as ports of entry for bacteria. It is good practice, therefore, to treat the wound again with some kind of antiseptic after all sutures have been taken out. In addition, the extraoral wound should be covered with a sterile dry pressure dressing.

SUMMARY

Careful and skilled removal of sutures will minimize the possibility of pain, wound separation, infection, and suture scarring. It has been observed

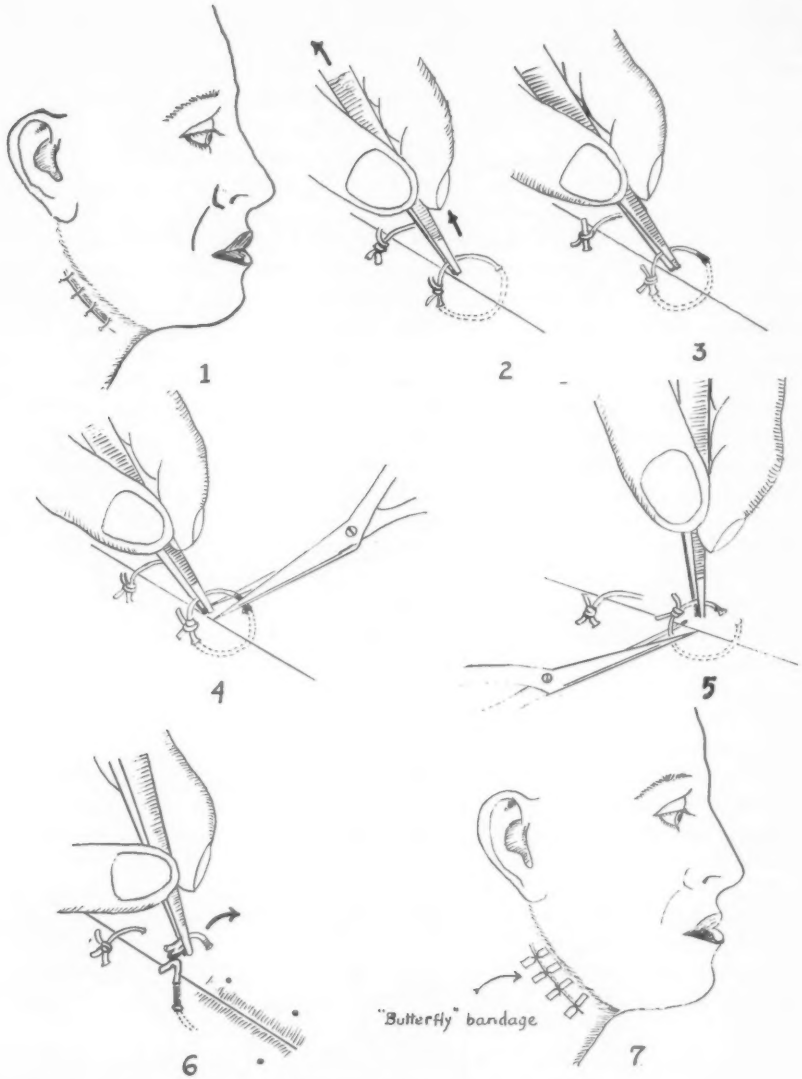

Fig. 1.—Extraoral wound with interrupted sutures.

Fig. 2.—Forceps grasp the extracutaneous part of the suture.

Fig. 3.—Gentle traction is applied only to buried portion.

Fig. 4.—Simultaneously the separated points of the scissors embrace the suture and slightly depress the tissue to expose a very short extent of the most subcutaneous part of the suture without drawing the extracutaneous part of the opposite side under the tissue. The suture is clipped close to the tissue.

Fig. 5.—Embracing the opposite part of the unremoved suture at its emergence from the tissue, the separated points of the scissors serve only to steady the tissue.

Fig. 6.—The submerged part of the suture is removed with the forceps, which still grasp the remaining extracutaneous portion and draw the suture toward the wound (see arrow).

Fig. 7.—The use of a "butterfly" bandage after all sutures have been removed is a further precaution to relieve all tension on the suture line.

that premature or hasty removal of sutures may lead to separation of the wound edges. On the other hand, if sutures are left in the wound too long, they will act as foreign bodies and cut through the skin tissue, increasing the amount of scarring. The technique of suture removal following oral surgery has been illustrated with diagrams.

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ALLERGIC HYPESTHESIA OF THE LIPS AND GINGIVAE

Report of a Case

Woodrow S. Monica, D.M.D., and Louis F. Raymond, M.D., Orange, N. J.

SUSPECTED allergic reactions sometimes cannot be confirmed for lack of substantiating evidence. A diagnosis may be made, however, if the allergy involves the nervous system of a patient who has had recurrent allergic attacks from ill-defined causes.[1]

In the following case, although the causes were defined, allergic hypesthesia of the second and third divisions of the trigeminal nerve is suggested.

CASE REPORT

In February, 1958, a 47-year-old dentist complained of numbness of the lips and anterior gingivae. The numbness lasted about two hours in the morning and was intermittent during the day for half-hour periods; it was accentuated by cold. The patient complained also of pressure in the ears, mild headache, pruritus of the penis and soles of the feet, and an annoying sensation in the gingivae. In addition, he noticed an increased rectal mucous secretion, a desire to empty his bowels, and abdominal pain. Basement odors made his nose "run." He took 1,000 mg. of vitamin C daily for three days, but the medication had no effect on the gingivae. Oral antihistamines did not lessen the hypesthesia.

The lips looked normal, but the anterior gingivae were moderately edematous. The mouth was free of any irritating causes, and dental roentgenograms showed no pathologic conditions. The maxillary sinuses were congested. Blood studies revealed normal red and white cell count and a normal concentration of hemoglobin. The differential blood count showed 7 per cent monocytes, 33 per cent neutrophils, and 60 per cent lymphocytes (the latter two in inverse proportion to normal blood counts).

The dentist was tested with intradermal injections of pollens and found to be sensitive to mixed tree pollens, giant and dwarf ragweed pollens, air-borne mold mixture, and newspaper allergenic extract. The results, read at intervals of six, twelve, and twenty-four hours, showed a four plus reaction (wheal with pseudopods) to tree pollens and two plus reactions to the remainder. There was no reaction to a control test with normal saline solution. The patient was desensitized over a six-month period by intradermal injections of allergenic extracts of the foregoing pollens in normal saline solution.

The patient was asymptomatic until February, 1959, when he again complained of slight intermittent numbness of the lips. He was desensitized again over a two-month period. Three months after the last injection, he was still symptomless.

COMMENT

Hypesthesia in the region of the second and third divisions of the trigeminal nerve is often seen in tabes dorsalis, cerebellopontine angle syndrome, occlusion

of the posterior inferior cerebellar artery, Sturge-Weber disease, benzene poisoning,[2] and allergic diseases.

In the second division of the nerve, hypesthesia is considered by some[3, 4] as an early sign of carcinoma of the maxillary sinus. Hypesthesia of the lips sometimes appears as an allergic reaction to food.[5] In hysterical patients, the hypesthesia does not always correspond to the organic nerve distribution and is influenced by suggestion and placebos.

Hypesthesia caused by a peripheral nerve lesion corresponds to the area of sensory distribution of the specific nerve.[2] The area of hypesthesia is usually smaller than the anatomic distribution of that nerve, and in the face it often crosses the midline.

Positive skin reactions are not necessarily of clinical significance unless corroborated by the case history. Some patients, free of allergic symptoms, may give positive skin reactions repeatedly for many years.

Although relief may follow hyposensitization, this does not mean that the condition was caused by an allergy. The relief may have been due to psychic satisfaction. In the case just presented, however, the condition was recurrent and hyposensitization did alleviate it on two occasions.

Allergic gingivitis occurs occasionally, but gingivitis with hypesthesia of the lips is uncommon.

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ORAL MEDICINE

SOME DENTAL MANIFESTATIONS OF MONGOLISM

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INTRODUCTION

THE appointment of one of us to a hospital for mental defectives led to the observation that the majority of Mongoloids among the patients appeared to be suffering from a characteristic periodontal condition which was much more severe than could be explained by local factors. It was also evident that their mouths were seriously involved at quite an early age.

What appear to be similar conditions have been reported by Benda[1] and Dow.[2] In his book, Benda reports the dental findings in eighty-four Mongoloid patients examined by Dr. John A. Nash. Their ages ranged from 3 years to 35 years, but more than half (forty-five) were between 6 and 15 years of age. The most significant observations were the relative freedom from caries, the frequency of paradentosis (seventy-eight cases), and abnormal alignment of the teeth (sixty-eight cases). Benda suggests that hypofunction of the gonads supplies an explanation. Dow described the periodontal disease that he observed as "periodontoclasia," which he defined as a general term for any degenerative or destructive disease involving the investing tissues supporting the teeth. Dow examined 151 "Mongolian type" children at the Polk State School. He found that from the age of about 3 years some periodontal disturbances were present, particularly flabby detached gingivae in the lower incisor region. It was not till the older age groups were examined, however, that calculus, inflammation, and destruction of the gingiva were found in the majority of mouths. The severity appeared to increase steadily with age, and about 50 per cent of the 13- to 18-year-olds had lost some teeth, while all the patients of 19 years and over had lost some teeth. Dow states that local factors could not explain the periodontal changes that he observed, and he suggests that anoxia due to poor circulation is a possible cause.

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In view of the above, we decided to make a survey of the dental condition of the eighty Mongoloid patients who, together with about 450 other mentally defective children, adolescents, and adults, are cared for in the Levin Farm Hospital.

The patients live in villas, each of which is designed to house a group of forty to fifty persons. The sexes are segregated and, with the exception of the infants and small boys, all age groups are represented in each villa.

There is a resident medical superintendent, and a registered psychiatric nurse is in charge of each villa.

Part of the establishment is a school for those able to take advantage of it, and there is a qualified occupational therapy staff. The grounds are very spacious, and games, sports, and other activities are encouraged.

TABLE I

AGE (YEARS)	NUMBER EXAMINED			
	MALE	FEMALE	TOTAL	PER CENT OF TOTAL
1-5	6	8	14	17
6-10	11	10	21	26
11-15	9	5	14	17
16-20	12	5	17	21
20-25	6	1	7	9
26 +	6	1	7	9
Total	50	30	80	

Table I shows the distribution of the eighty Mongoloid patients by age and sex. The youngest patient is 18 months old, and there are two others under the age of 3 years. The oldest patient is 39, and there is but one other patient over 30 years of age.

PROCEDURE

It was decided that the survey should record the prevalence of (1) periodontal disease, (2) malocclusion, (3) congenitally missing teeth, (4) calculus, and (5) dental caries. All the patients were examined by one of us (R. H. B.) in a well-equipped dental office, and a calibrated periodontal probe was used in addition to the usual mirror and probe.

Periodontal Disease.—The following were recorded: (1) pocket depth, in millimeters, (2) abnormal mobility, (3) ulceration of the gingiva, (4) bacteriologic report on a smear, (5) an assessment of the severity and extent of the periodontal involvement (the criteria suggested by Massler and Schour[3] were used), and (6) the color, consistency, and changes in the contour of the gingiva. The latter were used to amplify the assessment of the severity of the periodontal involvement, and, in addition, they provided a check on the validity of this assessment. *Gingival color* was recorded as pink, red, or bluish red. Gingival consistency was assessed as follows:

Normal. Stippled surface and firm to pressure.

Soft. Surface shiny and smooth due to absence of stippling; the bulk of the gingiva is firm, but the surface may pit in response to pressure from a ball burnisher.

Turgid. The bulk of the tissue is soggy due to inflammatory edema.

Changes in shape or contour of the interdental papillae and marginal gingivae were recorded as follows:

Interdental Papillae

Normal. Pointed, with a close approximation to the teeth.

Receded. Blunt or flat.

Everted. Marked loss of interproximal tissue without corresponding loss of labial and lingual tissue.

Enlarged. Papillae are bulbous and can be lifted away from the teeth.

Marginal Gingivae

Normal. The gingiva is firmly attached to the tooth surface and meets it in a thin edge.

Receded. The gingiva no longer covers the amelocemental junction.

Enlarged. The margins are thick and rounded and not closely adherent to the teeth.

The severity and extent of the periodontal involvement were assessed as follows:

0. No inflammation in any part of the periodontal tissues.

1. Mild gingivitis. Inflammation localized to the papillae and margins of the gingiva around one to three teeth.

2. Moderate gingivitis. Extension of the inflammation to involve the papillae and all the free gingiva around more than three teeth.

3. Severe gingivitis. Extension to involve the attached gingiva. Obvious edema, and tissue bleeds easily.

4. Generalized periodontitis. Abnormal mobility of the teeth and true pocket formation associated with a severe gingivitis.

Abnormal mobility was graded as follows:

1. Slight movement in excess of normal.

2. Movement in any direction not more than 0.5 mm. from the rest position of the tooth.

3. Movement beyond 0.5 mm. in any direction or teeth which can be rotated or depressed in their sockets. (''M3'' was considered ''gross'' mobility.)

Because of the number of patients showing some ulceration of the gingiva, it was decided that smears from the mouths of all patients should be taken. A platinum loop was used to collect material from the gingival crevice or a periodontal pocket. Where ulceration or acute inflammation was present, the smear was taken from the involved area. Smears were assessed as ''positive''

when large numbers of *Bacillus fusiformis* and *Borrelia vincentii* were seen, and there was a great predominance of these over all other types.[4] The smears assessed as "negative" showed none of these organisms. An "intermediate" state was noted where small numbers of *Bacillus fusiformis* and *Borrelia vincentii* were seen together with other organisms.

Malocclusion.—Under this general heading are included not only the three main classes of Angle's classification but also posterior cross-bite, open-bite, crowding of the anterior teeth, retained deciduous teeth, and evidence of a tongue-thrusting habit. Habitual lack of lip seal was recorded, but this is not necessarily evidence of mouth-breathing. The short period of observation did not justify recording other habits, such as lip-biting and thumb- and finger-sucking. Open-bite was recorded where there was a definite vertical space between the upper and lower incisors when the posterior teeth were in centric relation.

Missing Teeth.—Our interest here was in assessing how many patients had congenitally missing permanent teeth and whether some teeth were more likely to be missing than others. It was soon apparent that upper lateral incisors and lower incisors were frequently missing, and the loss was usually bilateral. The results reported are confined to the group of patients aged 8 to 13 years since in the older patients we were not able to exclude the possibility that missing teeth may have been lost because of periodontal disease.

As delayed eruption may have accounted for missing incisors among the 8- and 9-year-olds, all of these children were radiographed, with the exception of five who would not cooperate.

Calculus.—In normal patients there is a close association between periodontal disease and poor oral hygiene and the presence of calculus. Oral hygiene was assessed as "good," "fair," or "poor," and these findings were recorded separately for the anterior and posterior teeth. Supragingival calculus and subgingival calculus were recorded separately and graded as "slight," "moderate," or "severe."

Dental Caries.—Because of the early loss of teeth from periodontal disease, the congenital absence of some teeth, and the delay in the eruption of permanent teeth, an attempt at a DMF assessment was abandoned. However, the large number of "caries-free" dentitions was noted.

RESULTS

Periodontal Disease.—The prevalence of periodontal disease among male and female Mongoloids at various ages is shown in Table II. Three of the four patients with normal gingivae in the 1- to 5-year-old group are under 3 years of age. It is obvious that periodontal disease commences at an early stage in the majority of Mongoloid patients.

The severity of the periodontal involvement is shown in Table III. It will be noted that with age there is a progressive increase in the percentage of patients who have a generalized periodontitis.

TABLE II. PREVALENCE OF PERIODONTAL DISEASE BY AGE GROUPS AND SEX

AGE (YEARS)	SEX	NUMBER EXAMINED	NORMAL GINGIVAE	PERIODONTAL DISEASE
1-5	M	6	2	4
	F	8	2	6
Total		14	4 (29%)	10 (71%)
6-10	M	11	0	11
	F	10	0	10
Total		21	0 (0%)	21 (100%)
11-15	M	9	1	8
	F	5	1	4
Total		14	2 (14%)	12 (86%)
16-20	M	12	1	11
	F	5	0	5
Total		17	1 (6%)	16 (94%)
21-25	M	6	1	5
	F	1	0	1
Total		7	1 (14%)	6 (86%)
26 +	M	6	0	6
	F	1	0	1
Total		7	0 (0%)	7 (100%)
Total	M	50	5 (10%)	45 (90%)
	F	30	3 (10%)	27 (90%)
Total		80	8 (10%)	72 (90%)

All teeth were tested clinically for abnormal mobility, and the number of patients with one or more mobile teeth is shown in Table IV. A movement of more than 0.5 mm. in any direction (that is, a total translatory movement of

TABLE III. SEVERITY OF THE PERIODONTAL INVOLVEMENT BY AGE GROUPS

AGE (YEARS)	SEX	NUMBER EXAMINED	MILD GINGIVITIS	MODERATE GINGIVITIS	SEVERE GINGIVITIS	PERIO-DONTITIS
1-5	M	6	1	0	0	3
	F	8	3	0	1	2
Total		14	4 (29%)	0	1 (7%)	5 (36%)
6-10	M	11	1	1	2	7
	F	10	2	1	3	4
Total		21	3 (14%)	2 (10%)	5 (24%)	11 (52%)
11-15	M	9	1	1	1	5
	F	5	0	2	0	2
Total		14	1 (7%)	3 (21%)	1 (7%)	7 (50%)
16-20	M	12	1	1	1	8
	F	5	1	0	0	4
Total		17	2 (12%)	1 (6%)	1 (6%)	12 (71%)
21-25	M	6	0	0	1	4
	F	1	0	0	0	1
Total		7	0	0	1 (14%)	5 (71%)
26 +	M	6	0	0	0	6
	F	1	0	0	0	1
Total		7	0	0	0	7 (100%)
Total	M	50	4 (8%)	3 (6%)	5 (10%)	33 (66%)
Total	F	30	6 (20%)	3 (10%)	4 (13%)	14 (47%)
Grand Total		80	10 (13%)	6 (8%)	9 (11%)	47 (59%)

TABLE IV. ABNORMAL MOBILITY BY AGE GROUPS

AGE (YEARS)	NUMBER EXAMINED	ABNORMAL MOBILITY	"GROSS" MOBILITY
1- 5	14	5	5
6-10	21	16	5
11-15	14	11	6
16-20	17	15	10
21-25	7	5	4
26+	7	5	5
Total	80	57 (71%)	35 (44%)

1 mm. or more) was considered to be "gross" mobility, and the number of patients exhibiting such mobility is also shown.

At examination, a large number of patients showed some ulceration of the gingiva, and smears were made of all eighty patients. There were fifty-two (65 per cent) positive smears, nine patients (11 per cent) had no Vincent's organisms, and nineteen were assessed as intermediate. Ulceration was present in thirty-four patients (42 per cent), and all but one had a "positive" smear. The exception, a child with a congenital heart condition, had large numbers of mixed organisms, including masses of fungoid mycelium. As would be expected, there is a close correlation between the most severe periodontal involvement and the presence of a positive smear. This is shown in Table V.

TABLE V. PERIODONTAL DISEASE AND INFECTION WITH VINCENT'S ORGANISMS

PERIODONTAL ASSESSMENT	NUMBER EXAMINED	SMEAR			ULCERATION OF GINGIVA
		NEGATIVE	INTERMEDIATE	POSITIVE	
0 Normal	8	4	3	1	0
1 Mild gingivitis	10	1	4	5	0
2 Moderate gingivitis	6	0	4	2	1
3 Severe gingivitis	9	0	2	7	3
4 Periodontitis	47	4 (8%)	6 (13%)	37 (79%)	30
Total	80	9 (11%)	19 (24%)	52 (65%)	34 (42%)

The correlation between the over-all assessment of periodontal disease and the changes in the color, consistency, and contour of the gingiva is shown in Table VI. The heading "bleeding" refers to bleeding on palpation or during examination procedures (Table VI).

Malocclusion.—These patients have a high percentage of cross-bites in the posterior region. Twenty-two (71 per cent) of the thirty-one patients with posterior cross-bite also had a Class III malocclusion. There was no correlation between the degree of periodontal disease present and the type of occlusion. Table VII shows the types of occlusion present in the various age groups, and the total for each from 11 years old and older is given as well as the total for all age groups.

It is usually stated that Mongoloid patients have large tongues,[5-7] but Benda[1] states that the tongue tends to protrude because of the smallness of the oral cavity and not because of the size of the tongue. Either explanation would help to account for the high percentage of cases of open-bite, a tongue-thrusting habit, and lack of lip seal.

TABLE VI. CORRELATION BETWEEN OVER-ALL PERIODONTAL ASSESSMENT AND DETAILED FINDINGS

PERIODONTAL DISEASE ASSESSMENT	NUMBER EXAMINED	GINGIVAL COLOR			GINGIVAL CONSISTENCY				GINGIVAL CONTOUR							
									PAPILLAE				MARGINS			
		PINK	RED	BLUISH RED	NORMAL	SOFT	TURGID	BLEEDING	NORMAL	RECEDED	EVERTED	ENLARGED	NORMAL	RECEDED	ENLARGED	SPREAD OF INFLAMMATION TO ATTACHED GINGIVAE
0 Normal	8	8	0	0	8	0	0	0	8	0	0	0	8	0	0	0
1 Mild gingivitis	10	4	6	0	1	9	0	1	4	2	1	3	2	0	8	1
2 Moderate gingivitis	6	3	3	0	0	6	0	0	3	1	2	0	1	1	4	1
3 Severe gingivitis	9	0	7	2	0	9	0	5	1	2	4	2	0	2	9	6
4 Periodontitis	47	2	23	22	0	46	1	32	3	2	40	5	0	29	41	45
Total	80	17	39	24	9	70	1	38	19	7	47	10	11	32	62	53
Percentage	100	21	49	30	11	88	1	48	24	*	60	*	14	*	*	66

*These descriptions are not mutually exclusive; therefore, no percentages have been calculated.

Table VIII shows the prevalence of these habits, and the figures for open-bite are included in the same table.

Missing Teeth.—There were twenty-one patients in the 8- to 13-year-old group. Twelve of these had both permanent upper lateral incisors missing, but two 8-year-olds and three 9-year-olds could not be radiographed. If they are excluded, there are still seven (33 per cent) with both upper lateral incisors missing. There was one case in which one upper lateral incisor and two lower incisors were missing and the other upper lateral incisor was malformed. There was also

TABLE VII. OCCLUSION, ETC. AT VARIOUS AGES

AGE (YEARS)	NUMBER EXAMINED	CROWDED ANTERIOR TEETH	RETAINED DECIDUOUS TEETH	POSTERIOR CROSS-BITE	ANGLE'S CLASSIFICATION			
					NORMAL	CLASS I	CLASS II	CLASS III
1- 5	14	1	0	3	5	6	1	2
6-10	21	13	1	3	3	7	2	7
11-15	14	4	2	7	0	9	0	5
16-20	17	4	1	10	0	5	0	12
21-25	7	2	1	5	0	0	0	6
26+	7	0	0	3	1	0	0	4
Total from age 11	45	10	4	25	1	14	0	27
Percentage		22	9	56	2	33	0	64
Total all groups	80	24	5	31	9	27	3	36
Percentage		30	6	39	12	36	4	49

TABLE VIII

AGE (YEARS)	NUMBER EXAMINED	TONGUE-THRUSTING	LACK OF LIP SEAL	OPEN-BITE
1- 5	14	6	6	1
6-10	21	13	16	3
11-15	14	11	8	7
16-20	17	8	9	1
21-25	7	3	5	0
26+	7	1	4	0
Total	80	42	48	12
Percentage		52	60	15

a case in which the upper right lateral incisor was missing but not the left. Summarizing these findings, there were *at least* nine patients (43 per cent) in this age group who had one or both upper lateral incisors missing.

Six patients had bilateral loss of lower incisors. In four cases this was associated with bilateral loss of the upper lateral incisors and in one patient, as already mentioned, one upper lateral incisor was missing and the other one was malformed. In the sixth case both upper lateral incisors were present; this was, therefore, the only case of missing lower incisors without loss of one or both upper lateral incisors. This last patient should be ignored, since the lower incisors present were affected by advanced periodontal disease and it is possible that the missing lower incisors were lost due to periodontal disease even though the child was but 11 years of age.

Calculus, Etc.—Because periodontal disease in these patients appears to commence in the anterior regions and to be more severe here, the cleanliness of the mouth was assessed separately for the anterior and posterior regions.

TABLE IX. PERIODONTAL DISEASE IN RELATION TO ORAL CLEANLINESS

PERIODONTAL ASSESSMENT	NUMBER EXAMINED	ANTERIOR REGION			POSTERIOR REGION		
		GOOD	FAIR	POOR	GOOD	FAIR	POOR
0 No gingivitis	8	6	1	0	7	0	0
1 Mild gingivitis	10	1	9	0	9	1	0
2 Moderate gingivitis	6	1	4	1	3	3	0
3 Severe gingivitis	9	1	7	1	4	5	0
4 Periodontitis	47	2	18	26 (55%)	8	29	10 (21%)
Total	80	11	39	28	31	38	10
Percentage	100	14	50	35	39	49	13

Cleanliness or hygiene was rated as "good," "fair," or "poor." The proportion assessed as "fair" was the same for both the anterior and the posterior regions; however, 35 per cent of the mouths were rated as "poor," in the anterior region while only 13 per cent were rated as "poor" in the posterior region. For the patients with periodontitis, the comparable figures are 55 per cent "poor" in the anterior region and 21 per cent in the posterior region. The detailed assessments in relation to the periodontal disease present are shown in Table IX, and the same assessments in relation to age are shown in Table X.

TABLE X. ORAL CLEANLINESS IN AGE GROUPS

AGE (YEARS)	NUMBER EXAMINED	ANTERIOR REGION			POSTERIOR REGION		
		GOOD	FAIR	POOR	GOOD	FAIR	POOR
1- 5	14	4	7	3	10	3	1
6-10	21	3	12	6	10	10	1
11-15	14	1	10	3	6	8	0
16-20	17	3	5	9	5	11	1
21-25	7	1	3	3	1	3	3
26 +	7	0	2	4	0	3	4
Total	80	12	39	28	31	38	10
Percentage	100	15	50	35	39	48	12

Calculus formation, either supragingival or subgingival, is not a marked feature among these patients. More than half of the patients with periodontitis have neither type of calculus. Table XI shows the amount of calculus present in association with the degree of periodontal involvement. As would be expected, there is no calculus present in the "under 11" age groups, and the proportion without calculus decreases with age.

TABLE XI. CALCULUS FORMATION AND PERIODONTAL DISEASE

PERIODONTAL ASSESSMENT	NUMBER EXAMINED	SUPRAGINGIVAL				SUBGINGIVAL			
		NONE	SLIGHT	MODERATE	SEVERE	NONE	SLIGHT	MODERATE	SEVERE
0 No gingivitis	8	8	0	0	0	8	0	0	0
1 Mild gingivitis	10	10	0	0	0	10	0	0	0
2 Moderate gingivitis	6	6	0	0	0	6	0	0	0
3 Severe gingivitis	9	9	0	0	0	8	1	0	0
4 Periodontitis	47	25	18	3	1	34	9	1	3
Total	80	58	18	3	1	66	10	1	3
Percentage	100	72	23	4	1	83	13	1	4

Dental Caries.—These patients have a remarkable freedom from caries, particularly in the deciduous dentition. Table XII shows the number of caries-free dentitions in the various age groups. It should be noted, however, that some of the patients with caries-free dentitions in the older age groups have lost some teeth, and it is not possible to say whether these were lost to caries or to periodontal disease.

TABLE XII. CARIES-FREE DENTITIONS IN VARIOUS AGE GROUPS

AGE (YEARS)	NUMBER EXAMINED	CARIES-FREE DENTITIONS			TOTAL FOR AGE GROUP	GROUP PER CENT
		DECIDUOUS	MIXED	PERMANENT		
1- 5	14	13	1	0	14	100
6-10	21	3	7	0	10	48
11-15	14	0	0	2	2	14
16-20	17	0	1	6	7	41
21-25	7	0	0	0	0	0
26 +	7	0	0	2	2	29
All groups	80	16	9	10	35	44
To age 15	49	16	8	2	26	53

Freedom from caries in these patients does not appear to be related to oral cleanliness. The oral hygiene ratings for the caries-free patients are shown in Table XIII along with the percentages of caries-free dentitions, and dentitions that have been affected by caries are compared in terms of "good," "fair," and "poor" oral hygiene.

TABLE XIII. ORAL HYGIENE AND FREEDOM FROM CARIES

DENTITION	NUMBER CARIES-FREE	ANTERIOR TEETH			POSTERIOR TEETH		
		GOOD	FAIR	POOR	GOOD	FAIR	POOR
Deciduous	16	3	8	4	9	5	1
Mixed	9	1	6	2	5	4	0
Permanent	10	2	0	7	2	7	1
Total	35	6	14	13	16	16	2
Percentage		18	42	39	47	47	6
Total for dentitions affected by caries		5	25	15	15	22	8
Percentage		11	56	33	33	49	18

DISCUSSION

It has been shown that gingivitis is common in "normal" children[8-11] and that the severity increases until the age of about 15 or 16 years, but periodontitis or periodontosis is comparatively rare.[9] The results of this survey show that in Mongoloids periodontal disease commences at an early age, and in a very serious form, and that the alveolar bone is soon involved. The clinical features of a typical case are as follows: In a very young Mongoloid the first indication of the onset of the condition is a severe gingivitis, with ulceration and sloughing of the interdental papillae and gingival margins. In the young child (2 to 5 years of age) the condition is more acute than in the older victims; the child is irritable, with a slightly raised temperature, and the gingivae are painful, making feeding and oral hygiene procedures difficult. Local treatment, as for acute Vincent's infection, with 2 per cent chromic acid and full-strength hydrogen peroxide results in a rapid improvement but, partly because of the gingival damage and oral hygiene difficulties, the gingivae remain chronically inflamed. In the older patient, severe bone loss and gingival recession are the most marked features. The gingivae in the affected area are dark red in color, they are soggy due to inflammatory edema, they bleed on the slightest pressure, and the margins and interproximal area form a continuous band of ragged ulceration. False pockets are present, the gingival margins being flabby and lying away from the teeth. Tooth mobility is a noticeable feature, and exfoliation (particularly of lower incisors) is quite common. Oral hygiene problems are aggravated by the loss of interproximal tissue with the formation of retentive areas, by the false pockets present, and by blood and exudate forming a sticky medium for the retention of food. In the advanced case the condition does not appear to be painful, even though severe ulceration may be present and the child does not appear ill.

In looking for an explanation of our findings, the following features of Mongolism should be considered: abnormal circulation, susceptibility to infections, and specific alterations in the blood. Benda[1] states that there is

lymphopenia, and Hilliard and Kirman[12] report a "shift to the left" in the Arneth count (that is, the polymorphonuclear leukocytes tend to be immature). What is described as "abnormal circulation" is the combined effect of low blood pressure, a poorly developed vascular system (especially in the peripheral capillaries), and congenital heart defects. Probably, the well-known susceptibility to infections is due to cellular anoxia resulting from the abnormal circulation, to the abnormal leukocytes, and to the general endocrine insufficiency because the controlling influence of the pituitary gland is defective.[1] These same factors would also explain the poor repair potential of the soft tissues and the rapid loss of alveolar bone that is characteristic of these patients. Ulceration appears to be a likely result of any marked irritation of the gingiva, and large numbers of *Bacillus fusiformis* and *Borrelia vincentii* are present as secondary invaders. The ulceration does not spread widely, and other classic features of Vincent's infection are not present.

The possibility of a nutritional explanation[12] for the periodontal disease was considered, but the usual diet appeared quite adequate and vitamin and other supplements are regularly prescribed. Furthemore, patients who are not Mongoloids are receiving the same diet and they do not show advanced periodontal disease. Certainly, the diet is not a detergent one, but the nursing staff expends no little effort in cleaning the patients' mouths after meals. There is not a high correlation between periodontitis and poor oral hygiene and, indeed, the poor oral hygiene may be a result (due to tenderness of the gingiva, the presence of false pockets, and the loss of interdental papillae) rather than a cause of the periodontitis. It is also noteworthy that the presence of either supragingival or subgingival calculus does not appear to be a critical factor. All the periodontal disease reported by Benda[1] was classified by Nash as "periodontosis," and many of our patients show some of the typical features of periodontosis, such as abnormal mobility of the teeth, early and rapid loss of alveolar bone, lack of calculus formation, and a low caries incidence. However, it is usually considered that periodontosis does not occur before puberty in normal children, whereas in this group of Mongoloids severe gingivitis and even ulceration occurred quite frequently in the very young. It may be that further research into the pathology of Mongolism may throw some light on the etiology of periodontosis.

Local factors which may have some etiological significance in Mongoloids are exposure to air due to lack of lip seal and occlusal and muscular habits, such as tongue-thrusting.

Our examination of the occlusion supports the view that the maxilla is underdeveloped[1] while the mandible is more normal. Sixty-four per cent of the patients over 11 years of age had a class III occlusion, and almost all of these had a cross-bite in the posterior region. Our figures for missing teeth (43 per cent) cannot be compared directly with those of Dow and Nash since we restricted our observations to those in the 8- to 13-year age group where we could be sure that the teeth were congenitally absent. Dow[2] reported ten patients (40 per cent) out of a group of twenty-five in the group aged 8 to 12

years as having "teeth missing." Nash is reported by Benda[1] as having noted twelve cases (27 per cent) of missing teeth among forty-five patients aged 6 to 15 years. He stated: "It was not always possible to decide whether eruption was merely delayed or the teeth were completely missing. In several instances, X-ray examination was made and the absence of teeth definitely established."

Compared with the "normal" New Zealand child, these patients had a remarkable freedom from dental caries. Up to the age of 15 years, 53 per cent of the patients were caries-free when examined with a mirror and probe (bitewing radiographs were not taken). Considering the physical nature of the food served and the standard of oral hygiene that can be attained, this is an unusually low prevalence of dental caries. (The oral flora is being further investigated, particularly the number of lactobacilli and other acidogenic organisms.) Dow[2] found 33 per cent of his patients under 18 years of age to have "teeth decayed," and Nash is reported as having found 44 per cent of his patients with "moderate or severe caries."

SUMMARY

A survey was made of the dental conditions of eighty Mongoloid patients who are living in a hospital for mental defectives. Of the eighty, forty-nine were under the age of 15 years. The majority of the patients suffered from severe destructive periodontal disease which appears to commence at an early age, to attack the incisor regions, and to bear little relation to local etiological factors.

Calculus formation is not common, and there is no correlation between the state of oral cleanliness and the periodontal disease present.

About half the patients have a Class III occlusion (that is, the lower incisors are anterior to the upper incisors when the jaws are closed).

A high percentage of these patients have one or more congenitally missing teeth, and a surprising number show a complete absence of dental caries.

The findings of Nash, as reported by Benda,[1] and of Dow[2] are supported by this survey.

The authors wish to acknowledge the cooperation and assistance they have received from the Medical Superintendent, Dr. F. Cameron, and the staff of the Levin Farm Hospital. They are also grateful to the Department of Basic Sciences of the University of Otago Dental School for bacteriologic examinations.

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ORAL ROENTGENOLOGY

American Academy of Oral Roentgenology

Arthur H. Wuehrmann, Editor

RESEARCH OPPORTUNITIES IN TEACHING DENTAL RADIOLOGY

The Challenge of the Unknown

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THE development of competence in a researcher will not likely depend upon his participation in teaching, but almost invariably the effectiveness of a teacher will be enhanced by concomitant research activity. In the sciences and in the professions, education is characterized by limitations to the extent of knowledge which preclude full understanding of biologic or pathologic phenomena or the attainment of perfection in techniques. The teacher is thus beset by unsolved problems as he expounds the principles and the techniques of his field of interest. Rare indeed is the teacher who, through a desire to promote well-founded learning, is not impelled to seek some of the answers critical to his mission.

Within this context, research opportunities may be discerned for the teacher of radiology in dentistry. During the past fifteen years the complacency of earlier decades has yielded to one of the most exciting reappraisals of subject matter to occur within any teaching discipline. By no means confined to problems of radiation protection, the resurgence of investigative inquiry in oral radiology has doubtless been spurred by public demand for better understanding and control of radiation dosage factors. Because these involve almost every facet of diagnostic radiology, the upsurge of research activity has left few cherished principles unquestioned. Nor has interest been directed only to technical considerations, for it has become singularly important to justify the diagnostic necessity for each x-ray film in terms of an effective interpretation of tissue changes which it may portray.

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Thus, the teacher of radiology is unusually favored with possibilities for the initiation of research, in that much of his subject matter has become controversial. An atmosphere of urgency surrounds the solution of many of his problems, which often enough lend themselves to relatively small-scale study within manageable periods of time. Topics inviting attention, to suggest a few at random, include several of the factors which contribute to image density and contrast, the determination of limits beyond which power-input fluctuations and timer inaccuracy become critical for the resulting films, the emulsion characteristics of available x-ray films, the definition of diagnostically acceptable films in terms of their densitometric qualities, the development of still lower dosage techniques than have so far been devised, refinements in film-positioning technique to assure more consistently acceptable structural reproductions, the urgent need for new standards of film processing in or for dental offices, and the development of test phantoms valid for purposes of experimental investigation unlimited by considerations of patient dosage or availability. Many of these problems, it should be noted, can be resolved into elements compatible with the limitations and resources of small-scale studies; more extended types of investigative effort may be applied to probe further into the biologic implications of ionizing radiation or into challenging such possibilities as microradiography or the diagnostic use of radioisotopes.

Given, then, a curiosity to explore, the teacher will find research possibilities ranging from problems suited for limited personal investigation to more ambitious projects calling for a team of personnel backed by considerable material resources. From these he is able to elect a program of research commensurate with the circumstances in which he serves. He may content himself with utilizing some otherwise unscheduled time for the pursuit of limited studies on his own or with the collaboration of a colleague. He may seek to extend the scope of such investigations by recruiting the voluntary assistance of some of his students. The interest of graduate students (or of undergraduate students eligible for elective studies) may be directed toward specific projects which the teacher is anxious to sponsor and direct. The available full-time student summer fellowships offer exceptional opportunity for the promotion and direction of productive research activity. Finally, there is generous support available through governmental agencies for the larger-scale investigation of worth-while projects. By taking advantage of the opportunities which apply in individual circumstances, it is possible to devise a continuing program of research as an integral part of the activities of a teaching group in radiology. The problems which await investigation, joined by means for implementing their study, conspire to invite the attention of the teacher.

How may a program of research activities be expected to influence a teaching program? Ignoring, for the purposes of this article, familiar platitudes educed to support vague claims for the enrichment of teaching, let us examine some specific possibilities. The exhaustive collateral reading prerequisite to the initiation of research will inevitably develop a better-informed teacher, so closely attuned to the uncertainties in the body of knowledge which is his

subject matter that classroom dissertations will become more challenging for students. Comparably, the impact of his reading will permeate the seminars which have become essential to a well-rounded teaching program in radiology. And in the course of seminars it is noteworthy that "brainstorming" sessions can arise in which the keen young minds of students, newly encountering problems and unbound by any stereotyped approach, will sometimes originate surprisingly constructive suggestions.

Unresolved problems within the course content are nevertheless somewhat paradoxical in their potential teaching value. In drawing attention to their provocative stimulus to learning, I have not overlooked the fact that any realistic approach to teaching will also involve the provision of leadership and direction as the student progresses in his learning. Responding well to forceful and well-organized presentations, students are quick to sense indecision in their mentor. In the absence of clear frames of reference, confusion will prevail for students confronted with an overburden of unanswered problems in the subject matter. The challenge of the unknown will succeed best as motivation to learning only when it is introduced against a backdrop of clearly delineated principles and landmarks.

Ability to draw on personal accomplishments in research has a unique advantage accruing to any first-hand presentation—the presentation assumes a peculiar reality for the listener. This device is reinforced by a sense of conviction which the research-oriented teacher will unconsciously impart, so that students will tend to be the more impressed by his message. A word of caution is in order here: Personal research accomplishments should never be introduced unless they are directly pertinent to a discussion, and then only to an extent sufficient to illustrate or to fortify a point. Conceding the natural enthusiasm of any keen researcher for his own investigative work, this must be held in check within the classroom and structured to preserve the balance of more wide-ranging presentations. There is inherent danger that a labored intrusion of personal research into a discussion, verbally or through assigned readings, may submerge its potential import in quiet resentment of an apparent egotism. To underscore a message for the learner, personal research findings must be introduced judiciously.

Where the teacher's research findings have direct application in routine clinical technique, motivation for learning is achieved in a more subtle manner. Here the counterpart of first-hand verbal presentation is coupled with opportunity for the student to test the findings and to satisfy himself with their clinical significance. For the teacher, this proving ground contributes the ultimate sense of achievement; few experiences are so gratifying as the successful translation of research findings into clinical practice. Thus encouraged, the teacher acquires renewed vigor for continuing his research pursuits.

Research, no matter how well conceived, will seldom proceed without the inconvenient appearance of unforeseen variables and innumerable side issues. This is the almost inevitable experience of the investigator in the field of dental radiology, and it draws attention to yet another way in which research can

complement teaching. So many assumptions and misconceptions have, it seems, attended the scramble to research in radiology that the fabric of new knowledge is perforated with pitfalls and fallacies. These may be exemplified by persistent advocacy of the extended-distance technique as a prime instrument for dose reduction; by the intrusion into research of variables too often unrecognized or disregarded, to wit the frequent assumption that a primary beam will be of homogeneous intensity over the whole area exposed to its effects; by doubts cast on the "inverse-square" law through an insufficient consideration of anode locations in different tube heads; by misplaced confidence in film processing engendered by a considerable standardization of procedure; by assuming a complete transferability to clinical practice of wire mesh and stepwedge findings; by controversial interpretations of data on optimum kilovoltage levels, in regard to both dosage and contrast values; by disregard for anatomic limitations in otherwise well-conceived film exposure techniques; by experimentation based at times on unfortunate choices of instrumentation; and by radiation scatter patterns derived from wooden heads. These are but some disquieting aspects of the newer knowledge to which we should give pause; we may have accepted some concepts deserving of re-examination, either to elicit a better measure of support or to be discarded.

It is possible that there is a stern lesson for researcher and teacher alike. Eagerness to explore must never be given free rein unchecked by the hallmark of sound research procedure—an unceasing critical analysis of the methods, the data, and the interpretations involved in an investigation. The lesson is no less important for the student, for he must be taught how to evaluate the reports upon which he will rely, through the years, for his continuing education. The evaluation of research developments is a teaching responsibility with intriguing and constructive possibilities, whether applied informally in seminar discussions or through the assignment of prepared critiques.

The comments on the merit of some concepts developed through research will at least serve to forestall any naive belief that reliable and valid results will automatically accrue to plain hard work and conscientious effort. In actual fact, few research studies realize in full the hopes of their participants, particularly in terms of findings designed to have practical application. Should these emerge, there is, of course, good cause for gratification; irrespective of the outcome, however, there is always for the researcher a better understanding of the problem under study. If he also teaches, then in addition to some satisfaction of his quest for knowledge, he will acquire a broader if yet incomplete mastery of his topic which will serve well to back up discourses for those who learn with him.

Could it be that in the implication of this last phrase—a sharing of the learning experience—we discern teaching at its best? Unobtrusively, the teacher engaging in research may by his example provide the setting in which curiosity will take root. Dogmatism and complacency will be likely to yield before a spirit of inquiry that catches the imagination of students. It is motivation of this order which can vitalize a whole teaching program and produce a level of learning that is inconceivable under less favorable circumstances. And there is

a hint that the potential contribution of research in teaching will be partly unrealized if the activities must be conducted in physically separate locations. It will prove to be worth considerable inconvenience, both to efficient clinical teaching and to the uninterrupted pursuit of research, if provision can be made for teaching and research to be conducted within a common area. In large part, the inconvenience will stem from the seemingly ceaseless curiosity of by-standing students. This is all to the good, however, for it is evidence of spontaneous learning which is the more precious because it has not been spawned by any requirement in a school catalog.

Sharing of the learning experience finds its most direct expression through the actual inclusion of students in the research activities of their teachers. For the most part eager and willing to engage in research, students comprise a relatively untapped reservoir of human resources for those who direct research. However, the introduction of students to research requires careful planning, since much of their activity will consist of patient and time-consuming observation of an extremely routine order. While students will commonly need to be disillusioned of starry-eyed concepts of research as a vivid or dramatic occupational experience, this should never be attempted without bringing understanding into being as the illusion is dissipated. It is fundamental that in any project the relation of even the simplest and most routine tasks to the over-all objective should be well understood. When the import of each task is comprehended in relation to a whole plan of study, students can be counted upon to display all the qualities of patience and sustained interest necessary to the successful conduct of research. At times it will still be a neat trick to sustain the enthusiasm of students for research as they encounter its realities.

In a recent paper Phillips[1] has ably discussed several other aspects of student participation in research, including the careful selection of assignments for student responsibility, the students' background and training in relation to these assignments, their concurrent academic workload, and the concentration of activities for each student within limited areas. In summation, his timely discussion may be said to stress the need for good direction of student research efforts.

Reference should be made to another attribute which calls for special attention by those who direct student research participation. Experience suggests that meticulous and precise attention to detail, absolutely essential to research in radiology, will not develop by chance in present-day students. It has to be a function of good research direction to insist on this from the outset if disheartening results are not to ensue. A tendency to bypass detail has been observed repeatedly enough, but there is ample evidence that requirements for the detail of an investigation and its records can become a matter of personal pride to students properly indoctrinated in the methods and objectives of research. It is no disparagement of students to assert that good experience in this respect will not just happen. On the contrary, with such eventualities foreseen and countered by adequate preparation, the direction of student participation in research is an enviable experience for the teacher of radiology.

Tangible results may well attend the broader investigative scope permitted by the addition of student personnel, but any tangible results, no matter how impressive, will be overshadowed by the dramatic influence on a teaching program which attends the participation of students in research. It seems that an upsurge of interest arises and, extending into radiologic problems far beyond the immediate scope of an investigation, permeates from participants through whole classes of their fellow students. If the logic of this happy circumstance is obscure, it is certain that interest and imaginative thinking are aroused in a fashion unmatched by more conventional teaching devices. The experience of several schools collaborating in a recent radiation survey of dental offices will attest to this observation, as will numerous teachers whose privilege it has been to direct student research fellows assigned through the summer months.

At this point it is fitting to recognize that research, as a motivating force for learning, is not an exclusive monopoly of those teachers directly engaging in investigative pursuits. Not all good teachers are necessarily well suited to undertake original clinical or laboratory studies. This article has taken the view that effective teaching will depend in part upon a constant search for acceptable answers to unsolved problems. For some teachers, this will better take the form of appraising, correlating, and organizing the findings of others—seeking to piece together all the emerging new information in order to give clearer meaning to the whole. In this circumstance the motivation to learning inherent in research will be less readily evoked and will depend on deliberate didactic emphasis on the influence of research on the course content. However, the basic theme of this article—the challenge of the unknown in teaching—is not weakened by admission that an active and direct participation in research is not the sole outlet for an inquiring mind.

If, through reiteration of the influence of research on teaching, there has been digression from the topic assigned, it has beeen in the belief that the objectives and potentials of research in teaching are less evident than are the opportunities for it. It is further admitted that there has been no attempt to conceal personal enthusiasm for the integration of research activity into careers in dental education. With due allowance for personal bias, it is nevertheless safe to submit that the conduct of research satisfies the natural drives of the true teacher as it serves to sustain his interest; for students it initiates a contagious experience in learning, engendering a habit of curiosity and a spirit of critical inquiry in direct proportion to the extent that the experience can be shared. A whole teaching program can become alive as teacher and students alike reach for understandings. Through research opportunities in radiology there is, indeed, an ideal motivation for real learning. For what more could any teacher hope?

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CALCIFICATIONS OF THE DENTINAL PAPILLA THAT MAY CAUSE ANOMALIES OF THE ROOTS OF TEETH

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THIS report concerns roentgenographic observations of abnormal calcification of the dentinal papilla which starts after the crown of the tooth has formed and which may cause deformity and abnormality of the root. The degree of deformity of the root is dependent upon the degree and extent of the calcification. The crowns of involved teeth are normal in shape, size, and color. There is a tendency to multiple occurrence, and in a given case several or possibly all of the teeth may be involved. In this report we are presenting our roentgenographic observations in three cases of abnormal calcification of the dentinal papilla. Since such calcification is more clearly demonstrable in teeth with single roots than in the others, roentgenograms of premolars and canines have been selected from three cases to illustrate variations in the size and position of the calcified nodules.

ROENTGENOGRAPHIC OBSERVATIONS IN THREE CASES

That the calcification can occur early in life is illustrated by Fig. 1. The patient was a girl, 13 years of age. The calcified nodules had caused little, if any, abnormality of the roots. All of the teeth were present, and roentgenographic evidence indicated that at least nine of the teeth were involved. On viewing the roentgenogram, one sees first the round roentgenopaque mass or nodule that occupies the center of the root. The nodule appears to be free and is not attached to the pulpal wall, for a narrow roentgenolucent line which depicts the pulpal space is continuous around it and separates it from the dentine. Also, the nodule remains separated from, and does not become fused with, the calcified material in the pulp chamber which extends into the pulp canal and comes in close proximity to it. This is evidenced by a distinct horizontal roentgenolucent line which separates them and which can be seen in this case, especially in the canine and first premolar teeth. The dentine of the root is continuous with the dentine of the crown, but it is abnormally thin where it surrounds the calcified mass in the center of the root. The apical portion of the root is normal in shape and size and contains a normal root canal that terminates at the apex. The periodontal membrane space and surrounding bone have a normal roentgenographic appearance.

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Dental roentgenograms (Fig. 2) made for a 55-year-old man revealed calcified nodules that were very similar to those shown in Fig. 1. These were found in ten of the twenty-five teeth present. Roentgenograms of the four mandibular premolars are illustrated in Fig. 2. The patient had not had any symptoms referable to the nodules, and the teeth gave a normal response to tests for vitality.

Fig. 1.—Calcified nodules in the root canals of the canine and premolars of a 13-year-old girl. These presumably formed prior to completion of development of the roots. (Courtesy of Drs. T. R. Abbott and L. E. Hinz, Milwaukee, Wisconsin.)

Fig. 2.—Calcified nodules which occupy a large pulpal space in the root canals of the mandibular premolars of a 55-year-old man.

Apparently, in these two cases calcification of the dentinal papilla was not so extensive that it had interfered with the formation of a root of normal size and form, although slight bulging of the root may have been present opposite the calcification in some of the affected teeth.

Deformity of the root may result when calcification of the dentinal papilla is more extensive. The root may have a bulbous shape, and in some cases the root of a single-rooted tooth will be enlarged to the extent that its diameter will exceed that of the crown of the tooth. The apical portion of the root is shortened, or it may be narrow and spindle-shaped. The roentgenogram made for a 35-year-old woman illustrates deformity of the roots (Fig. 3). The roentgenopaque mass which occupies the central part of the root in the canine

and premolars is surrounded by a layer of dentine which has grown around the obstacle, thereby enlarging the roots and giving them a bulbous shape. The apical portion of the root of the second premolar is short, and that of the canine and first premolars is narrow and spindle-shaped. Here again, the periodontal membrane space and the surrounding bone are roentgenographically normal. The root canal which leads to the apex of the root is very narrow, but it is discernible in the original roentgenogram, as is a roentgenolucent line around the nodule which separates it from the pulpal wall. While no definite nodules are discernible roentgenographically in the second and third molars, their roots are short and blunt, as were those of the other molars present.

Fig. 3.—Large calcified nodules in the root canals, which have caused deformity of the roots of the canine and premolars.

COMMENT

Hoggins and Marsland[2] reported a case in which the roots of twenty-three teeth that were extracted from a 30-year-old man were morphologically abnormal. Roentgenograms made of three of the extracted teeth are strikingly similar to those shown in Fig. 3. On microscopic examination, Hoggins and Marsland found that the mass within the root consisted chiefly of small foci of calcific degeneration and dentinal tubuli of irregular pattern running parallel to the long axis of the tooth. The mass was surrounded by a thin layer of normal dentine. An interesting feature of their report was that the patient was suffering from calcinosis universalis which first was recognized when he was 7 years of age. They attributed the abnormality of dental development to this condition. In the three cases to which we have referred, however, the calcification of the dentinal papilla was apparently not associated with any particular systemic condition, and there was no history of metabolic disturbance or generalized skeletal disease. Dental anomalies frequently follow a familial or hereditary pattern, but unfortunately no information was available that would substantiate or rule out this possibility in our cases.

From the location of the nodules, it must be assumed that they were formed shortly after the crown developed and that they had reached some size before an appreciable amount of the root had developed. This is evidenced by the

abnormally large pulpal space occupied by the nodule. Calcification occurring in the pulp later in life does not cause resorption of the pulpal wall to produce such enlargements of the root canal; nor does it produce a bulging of the outer surface of the root.

The early formation of denticles is in itself not a rarity, for denticles have been found in tooth germs before eruption.[1] According to Johnson and Bevelander[3] and others, denticles are mostly confined to the pulp chamber, and calcification of the pulp in the root canal is diffuse. The roentgenopaque objects seen in the root canals of some of the teeth in the cases reported here were single calcified nodules which did not resemble the diffuse calcification commonly seen in that region. Although so-called true denticles occur only rarely, Orban[4] has observed that they are usually found close to the apical foramen. The fact that the nodules referred to in this report did not tend to fuse with other areas of calcification that were present in the pulp suggests that they may consist chiefly of dentine.

Since calcification of the dentinal papilla occurs prior to eruption of the teeth, attrition, erosion, and caries to which calcifications of the pulp are often attributed cannot be causative factors. Almost all of the teeth involved in our cases had been and still were free from caries, and the few restorations that were present were not extensive. There was no apical infection or cyst formation. Apparently, interference with the normal function of the odontoblastic layer had not occurred; therefore, the continuity of the pulp from the chamber to the apex of the root had not been interrupted, and the teeth had remained vital. Whether a more marked calcific degeneration of the papilla would interfere with normal odontoblastic activity and result in dentinal dysplasia with obliteration of the root canal is problematic.

This report on calcification of the dentinal papilla is based on roentgenographic observations only. The condition is relatively rare. We are submitting it for publication in the hope that others who encounter similar cases may have an opportunity to make microscopic examinations in order to learn the true nature of the calcification and also that they may be able to ascertain whether or not the condition has a familial distribution.

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ROENTGENO-QUESTIONS

Is my timer (mechanical) accurate for exposures of less than one second?

The current trend toward reducing the exposure of dental patients to x-radiation through the use of high-speed films has created technical problems for the owners of x-ray machines equipped with mechanical timers. Many of these timers are inaccurate below one second; thus, they may yield varying exposure times even with identical settings. The length of time required to heat the filament of the tube also becomes a significant factor in the exposure of films that require exposure times of less than one-fourth second.

There are various means which permit the exposing of high-speed films for intervals of time which can be controlled with sufficient accuracy by mechanical timers. If a given high-speed film requires an exposure of 2.5 milliampere-seconds, this can be achieved with an exposure of one-fourth second with 10 Ma.; this may not be possible with the mechanical timer. The exposure also can be achieved in one and one-fourth seconds with 2 Ma. This period of time can be controlled successfully with the mechanical timer. Whether or not a given x-ray machine can be adjusted to operate with 2 Ma. and, if so, how this may change the voltage which is applied to the x-ray tube can be learned from the manufacturer of the apparatus.

The use of additional filtration and the use of a greater focal-film distance are additional methods of prolonging exposure times to the point where they can be controlled adequately with mechanical timers.

Accurate electrically operated timers are available as replacements for mechanical timers. On suitable x-ray machines, these timers have been very successful.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

A Case History

THE accompanying roentgenograms were received for publication in "Roentgeno-Oddities." They were accompanied by a rather sketchy history which indicated that a skeletal roentgenographic survey (including films of the spine, long bones, ribs, and skull) showed a suggestion of dense ossification but no clear-cut abnormality. Some hereditary tendencies may exist, since there is a history of other members of the patient's family having had their teeth removed at a very early age. The patient appears to be a healthy man in all respects. Results of clinical laboratory tests (the extent of which is not known) were within normal limits.

It was realized, of course, that a diagnosis should not be made on the basis of the roentgenographic films alone, but the editor felt that some speculation about these films might be of interest to the readers of the JOURNAL. An opinion was solicited from Dr. Edward Stafne, who comments as follows:

Judging from the dental films, the report of the roentgenologist, and the limited history, there are two conditions that immediately come to mind: amelogenesis imperfecta and osteopetrosis.

Amelogenesis imperfecta could no doubt be diagnosed on the basis of the clinical examination, but the x-ray findings are important in that they suggest normal dentine and a normal pulp cavity. These findings imply that we are dealing primarily with an ectodermal defect. The pulp cavities may be slightly larger than average, but they may appear enlarged because of the absence of a normal thickness of enamel on the surface. Since this particular defect has a familial tendency, it is interesting to learn that a sister of the patient had lost her teeth at 18 years of age. One could almost read between the lines and assume that the crowns of this young lady's teeth were tapered, had a horrid brownish color, and were extracted for that reason.

Osteopetrosis (Albers-Schönberg disease, marble bones) is something else again. The most widely accepted view is that the fundamental defect in osteopetrosis is a faulty differentiation of the hematogenic and osteogenic tissues. Both arise primarily from undifferentiated mesenchyme; therefore, this disturbance should have no effect upon the develop-

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Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

ment of enamel. However, the dental films show a homogeneous opacity of the bones of the maxilla and mandible and suggest that the maxillary sinuses are very small. Both findings are consistent with osteopetrosis. The sinuses are often small and sometimes obliterated, and the general roentgenologist probably had this in mind when he requested further x-rays of the sinuses. In his report he states that there is "no clear-cut abnormality," and here he no doubt refers to the configuration of the bones. This statement is significant, for in osteopetrosis the bones usually retain their normal contour. Clubbing of the epiphyses of the long bones is an exception, but this is not always present in the benign cases. His reference to "radiodensity" and "dense ossification" of the many bones that he has already examined may be sufficient evidence to establish a diagnosis. Many of the cases reported in the literature are based on the roentgenologic examination per se. Since the condition has a marked familial tendency, it is often further substantiated by similar findings in other members of the family. It would be interesting to have roentgenograms of the jaws and skull of the patient's sister. The base of the skull is sclerosed in almost all cases.

The case in question would have to be the adult benign form of the disease, the one which is compatible with normal living and which may be asymptomatic or only mildly symptomatic. Usually this form is first recognized incidental to roentgenologic examination for some other reason—perhaps, in this case, incidental to the dental examination. It is very important that it be differentiated from the malignant childhood form of this disease.

As for differential diagnosis, there are several forms of osteosclerosis, but most of them are confined to one or a few bones and are not generalized as is the case in question. Among other generalized forms are those associated with blood dyscrasias. Blood dyscrasia can be eliminated, since the results of the clinical laboratory tests are normal.

Going back to the dental films, the periapical lesions on the lower incisors have been noted. Their appearance suggests granulomas. In view of the relatively high incidence of osteomyelitis in osteopetrosis (if it is that), it would be well to make use of antibiotics when and if the teeth are extracted.

In conclusion, let me suggest two articles on osteopetrosis that have proved to be of considerable value:

McCune, D. J., and Bradley, C.: Osteopetrosis (Marble Bones) in Infant, Am. J. Dis. Child. 48: 949-1000, 1934.

Piatt, A. D., Erhard, G. A., and Araj, J. S.: Benign Osteopetrosis, Am. J. Roentgenol. 76: 1110-1131, 1956.

ORAL PATHOLOGY

General Section

ORAL LYMPHOMAS

H. P. Cook, M.B., B.S., M.D.S., F.D.S.R.C.S., London, England

LYMPHOMAS are malignant neoplasms of lymphoid tissue which in the past were often described under the collective name of reticuloses. It is now widely known that these rare conditions can occur in the mouth as apparently solitary lesions.

In this article an attempt will be made to delineate more clearly the characteristics of these tumors, so that their clinical features may be more readily recognized by dental workers.

An oral lesion can be the first to cause symptoms, although it may be one of many that are present throughout the body. On the other hand, an oral lesion may be the only one evident when the patient is first seen and precede involvement of the lymph nodes, spleen, or liver by varying periods. There is also the special case of primary reticulum-cell sarcoma of bone, first described separately by Parker and Jackson,[24] in which generalized disease may follow discovery of the initial lesion by many years.

There have been a few reviews of this subject based upon the analysis of cases treated at various centers. At the Mayo Clinic Steg, Dahlin, and Gores[30] made a statistical review of cases in which malignant lymphomas occurred in the jaws. They accepted forty-seven cases, all with histology, out of a possible eighty-two in this category. Of these tumors, ten were lymphosarcomas, fourteen were reticulum-cell sarcomas, two were Hodgkin's disease, and twenty-one were transitional types. All were regarded as primary tumors occurring in bone, although enlarged cervical nodes were present in twenty-three cases. The most common presenting symptoms were pain and swelling. Unfortunately, the period of time over which these cases were seen is not given, so it is not possible to form any estimate of frequency; however, the relative incidence of the various types is brought out.

Mayo Clinic material was also used in a review of primary reticulum-cell sarcoma of bone by McCormack, Ivins, and Dahlin.[20] They found thirty-two

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cases of this condition, but the jaws were not affected in any of them. In a later paper, Ivins and Dahlin[16] presented forty-nine cases of the condition. In this series the mandible was the site of disease in two cases and the maxilla in one.

Coley, Higinbotham, and Groesbeck,[5] reviewing thirty-seven Memorial Hospital cases of primary reticulum-cell sarcoma of bone, found no case in which the jaws were affected.

Nine cases at sites other than the jaws were reported by Medill,[21] but this author also stated that out of 121 cases found in the literature, four were reported as affecting the mandible. Gerry and Williams[11] reported two cases in which the mandible was involved.

Obviously, primary reticulum-cell sarcoma of bone is a rare tumor; Lumb[19] cites three out of twenty cases of reticulum-cell sarcoma in the Westminster Hospital series of 410 malignant lymphomas seen between 1940 and 1952. For this tumor to occur in the jaws must be very rare indeed.

Most of the other cases of malignant lymphoma of the mouth reported in the literature consist of single cases (Table I). All those included had satisfactory histology.

These thirty-four cases are analyzed according to site in Table II.

An additional six cases of lymphoma in the mouth seen between 1952 and 1959 will be described. Only cases in which the jaws and related soft tissues were involved have been included. Cases in which the tumors were located in the tonsils and pharynx have been omitted, as these sites are not the concern of the dental surgeon.

As far as terminology is concerned, Lumb's classification will be used. Lumb has formulated a classification of malignant lymphomas based on differentiation of the predominant cell type. Thus, if the cell types are fundamentally derived from primitive mesenchymal cells which are pluripotential, it is possible for them to give rise to tumors in which lymphocytes predominate, tumors in which reticulum cells predominate, and tumors in which mixed cell proliferation is present. Within each group there occur variants of each main type of different anaplasia, and these carry a roughly corresponding difference in prognosis. The types recognized are as follows:

1. Those showing lymphocytic differentiation
   (a) Lymphosarcoma, including lymphatic leukemia presenting as lymphadenopathy
   (b) Follicular lymphoma
2. Those showing reticulum-cell differentiation
   (a) Reticulum-cell sarcoma
3. Those showing mixed cell types
   (a) Hodgkin's disease
   (b) Reticular lymphoma
4. Very anaplastic types making cell-type differentiation impossible
   (a) Anaplastic sarcoma of lymphoid tissue

TABLE I

AUTHOR	DISEASE	SITE	REMARKS
Calman[3]	Lymphosarcoma	Left maxilla	17-year-old boy; numbness in left upper lip and swelling, 3½ months; histology; first seen by dentist
Salman and Darlington[27]	Lymphosarcoma	Right maxilla	7½-year-old girl; swelling, 7 months; histology; first treated by dentist
Hecht[14]	Lymphosarcoma	Left maxilla	23-year-old man; pain and swelling, 3 months; nodes and chest involved; histology
Freedman[10]	Lymphosarcoma	Palate	54-year-old man; pain and swelling, 3 months; histology; treated by radiotherapy; clear 15 months later
Fanale and McCauley[8]	Lymphosarcoma	Palate	69-year-old man; swelling; nodes present; histology
Thoma[32]	Lymphosarcoma	Palate	33-year-old woman; swelling; nodes and chest involved; histology
Silverman[29]	Lymphosarcoma Lymphatic leukemia (?)	Upper gingiva From $\underline{4}\vert$ to $\vert\underline{4}$	48-year-old woman; gingival swelling; blood count—W.B.C. 16,600, lymphocytes 33%; later developed nodes and breast lump; histology; died 5 months after diagnosis
Orsós[23]	Lymphosarcoma Lymphatic leukemia (?)	Gingivae Generalized	7-year-old girl; gingival swelling; histology; blood count—W.B.C. 4,000 c.mm., lymphocytes 50%, and 4% atypical forms
Burstone and Baetz[2]	Lymphosarcoma	Right mandible	67-year-old woman; pain and swelling, 7 months; nodes present; histology; radiotherapy; remission
Rollins and Thoma[26]	Lymphosarcoma	Left mandible	44-year-old man; swelling and numbness of lip, 1 month; histology; radiotherapy
Christiansen[4]	Lymphosarcoma	Maxilla and mandible	5-year-old boy; swelling, 2 months; death, 2 months; histology
Thoma[32]	Lymphosarcoma	Maxilla and mandible	66-year-old man; swelling; histology
Seldin, Seldin, and Rackower[28]	Lymphosarcoma	Cheek and left upper alveolus	64-year-old man; pain and swelling, 8 days; histology; death, 2 months
	Lymphosarcoma	Right mandible	29-year-old woman; swelling, 4 months; histology
Seldin, Seldin, and Rackower[28]	Reticulum-cell sarcoma	Right maxilla	73-year-old woman; swelling, 4 months; histology
	Reticulum-cell sarcoma	Right maxilla	54-year-old man; swelling, 7 months; nodes present; histology; radiotherapy; death, 5 months
Kennedy[17]	Reticulum-cell sarcoma	Right maxilla	80-year-old woman; swelling and necrosis; histology; radiotherapy; death rapid
Thoma[32]	Reticulum-cell sarcoma	Left maxilla, alveolus and sulcus	63-year-old man; swelling and ulceration; histology
Bernier[1]	Reticulum-cell sarcoma	Alveolus and palate	4-year-old boy; pain and ulceration; 1 month; histology; treated by dentist first

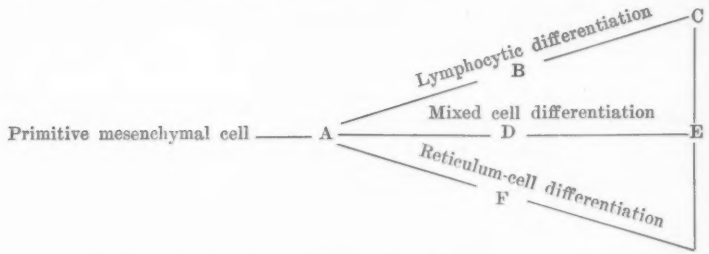
TABLE I—CONT'D

AUTHOR	DISEASE	SITE	REMARKS
Goldman[12]	Reticulum-cell sarcoma	Palate and soft palate	24-year-old man; swelling and pain, 1 month; treated by dentist first; histology; radiotherapy; good response
Harrigan, Butler, and Schaffer[13]	Reticulum-cell sarcoma	Left mandible	70-year-old woman; preceded by osteomyelitis; swelling, 2 years; swelling and numbness of lip; surgical excision; 3-year cure; histology
Penhale[25]	Reticulum-cell sarcoma	Right mandible	28-year-old man; pain and swelling, 3 months; numbness of lip; histology; radiotherapy; death
Frank and Pratt[9]	Reticulum-cell sarcoma	Left mandible	42-year-old woman; pain and lip numbness, 3 months; treated by dentist; histology; metastases
Ivins and Dahlin[16]	Primary reticulum-cell sarcoma of bone	Maxilla	No details
		Mandible	68-year-old woman; painless lump, 7 months; histology; 5-year survival
		Mandible	9-year-old boy; painless lump, 4 months; histology; 8-month follow-up
Gerry and Williams[11]	Primary reticulum-cell sarcoma of bone	Mandible	22-year-old man; painful lump and numbness of lip, 6 months; histology, 22-month follow-up
		Mandible	19-year-old man; painless lump, 6 months; histology; 11-month follow-up
Khanolkar[18]	Primary reticulum-cell sarcoma of bone	Mandible	20-year-old man; painless lump, 4 months; histology
Parker and Jackson[24]	Primary reticulum-cell sarcoma of bone	Mandible	14-year-old boy; painless lump, 3 months; histology; 4-month follow-up
Szutu and Hsieh[31]	Primary reticulum-cell sarcoma of bone	Mandible	59-year-old woman; painful lump, 3 months; histology; 1-year follow-up
Helling[15]	Primary reticulum-cell sarcoma of bone	Mandible	5-year-old girl; lump; histology; 10-year follow-up
Eisenbud and Kotch[7]	Hodgkin's disease	Cheek	55-year-old man; painless swelling; known case of Hodgkin's disease; enlargement of nodes and spleen; histology
Meyer, Rosmit, and Ungers[22]	Hodgkin's disease	Right mandible	33-year-old man; pain and failure to heal, $\frac{2}{8}$; nodes in neck; histology; radiotherapy; well 1 year

TABLE II

TUMOR	MAXILLA	MANDIBLE	BOTH	GINGIVAE	TOTAL
Lymphosarcoma	7	3	2	2	14
Reticulum-cell sarcoma	6	3	–	–	9
Primary reticulum-cell sarcoma of bone	1	8	–	–	9
Hodgkin's disease	1	1	–	–	2
All lymphomas	15	15	2	2	34

The concept has been well illustrated diagrammatically as follows:



Histogenesis of tumors of lymphoid tissue. This is diagrammatic and does not imply that better-differentiated types are derived from anaplastic types.
A = Lymphoblastic reticulosarcoma, anaplastic sarcoma of lymphoid tissue, and anaplastic reticulum-cell sarcoma.
B = Lymphosarcoma and lymphatic leukemia.
C = Follicular lymphoma.
D = Hodgkin's disease.
E = Reticular lymphoma.
F = Reticulum-cell sarcoma.
(Reproduced by permission of E. & S. Livingstone, London.)

The cases comprising the present series are represented in Table III.

TABLE III

TYPE	NUMBER	SITE
1. Lymphosarcoma	1	Upper alveolus central incisor region
2. Lymphatic leukemia	1	Lower alveolus left first and second molar region
3. Follicular lymphoma	1	Left buccal mucosa and cheek
4. Reticulum-cell sarcoma	1	Upper lip
5. Reticular lymphoma	1	Lips and buccal mucosa
6. Anaplastic sarcoma of lymphoid tissue	1	Lower alveolus, left canine region

CASE REPORTS

CASE 1. LYMPHOSARCOMA.—A 61-year-old man (a journalist) complained of pain on brushing the upper incisors. This had been present for six weeks. At about the same time he noticed a swelling under his upper lip which gradually enlarged and caused considerable discomfort. It had not ulcerated or bled. The upper incisors were extracted and a biopsy was performed. The biopsy report indicated a chronic inflammatory process. The patient was referred to a special center about one week later.

Examination revealed a mass of about 5 by 3 cm. occupying the anterior part of the upper alveolus, from which the central incisors were absent. The surface was smooth, and the consistency was hard to rubbery. The mass was fixed to the deep structures and extended on to the palate (Fig. 1). One rubbery mobile node was felt in the submandibular region, and moderate axillary glands were present. The liver and spleen were not felt.

Roentgenographic investigation showed invasive destruction of the anterior part of the maxilla (Fig. 2). The chest was clear.

Blood investigation disclosed the following: hemoglobin 103 per cent; white blood cells, 9,000 per cubic millimeter; lymphocytes, 7 per cent; polymorphonuclear leukocytes, 81 per cent; and monocytes, 10 per cent.

Biopsy showed a "diffuse infiltration by cells of lymphocytic type invading widely; regarded as a lymphosarcoma" (Fig. 3).

The tumor and node responded well to radiotherapy, and the patient is well and free from disease five years later.

Comment.—In this patient pain and swelling were the first symptoms. The condition was not correctly diagnosed at first, teeth were extracted, and the first biopsy was noninformative. The lesion appeared to be involving bone.

Fig. 1.

Fig. 2.

Fig. 1.—Case 1. Lymphosarcoma. Upper incisor region showing lesion.
Fig. 2.—Case 1. Roentgenogram showing bony destruction of alveolus in upper incisor region.

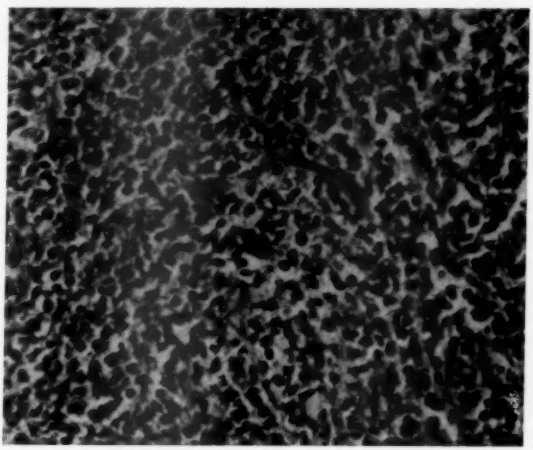


Fig. 3.—Case 1. Histology. Uniform arrangement of lymphocytes indicative of lymphosarcoma. (Hematoxylin and eosin stain. Magnification, ×240; reduced ⅓.)

CASE 2. LYMPHATIC LEUKEMIA.—A 40-year-old housewife noticed a painless swelling on the outer surface of the left lower alveolus about four months previously. It had not bled or ulcerated, and she had no general symptoms. About two months later a dentist extracted the left lower second molar, but this had no effect on the swelling.

On examination, a firm nonulcerated swelling was found in the molar region of the left lower buccal sulcus. It extended over the alveolar ridge and was fixed to the underlying bone. Externally, a lump could be felt on the outer aspect of the left mandible in relation to the intraoral lesion. No enlarged lymph nodes were present. Roentgenograms showed no bony abnormality.

A biopsy was performed and reported (Fig. 4) as follows: "Shows a type of lymphoid tissue in which there are definite germ centers in some areas, while in others the cell types are mixed. Thought to be a fairly well-differentiated follicular lymphosarcoma." At that time the blood count was normal with 8,000 white blood cells per cubic millimeter, of which 6 per cent were lymphocytes, 89 per cent were polymorphonuclear leukocytes, and 5 per cent were monocytes. The lesion disappeared with radiotherapy, and the patient remained well for two years and eight months.

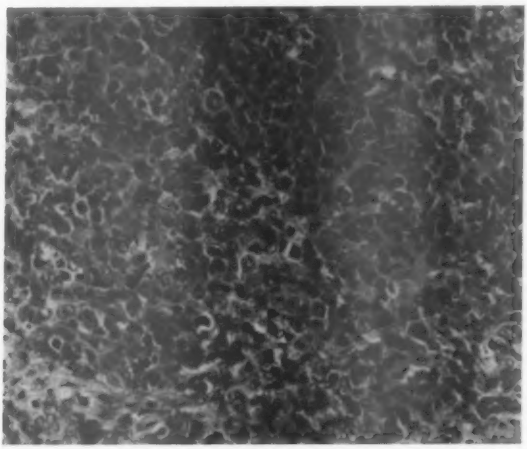


Fig. 4.—Case 2. Histology. Fairly well-differentiated lymphocytes suggesting lymphosarcoma. (Hematoxylin and eosin stain. Magnification, ×240; reduced ⅛.)

She then returned with widespread lymphadenopathy involving the cervical, axillary, and inguinal groups. The spleen was enlarged to the level of the umbilicus, and the liver was enlarged two fingerbreadths below the right costal margin. Blood findings were as follows: white blood cells, 18,200 per cubic millimeter; lymphocytes, 59 per cent; "blast" cells, 22 per cent; polymorphonuclear leukocytes, 12 per cent; and monocytes, 4 per cent. Sternal marrow showed: "Total nucleated count 120,000 per cubic millimeter, an excess of abnormal small lymphoid cells with normal marrow cells in the background. Suggests dissemination of lymphosarcoma rather than classic lymphatic leukemia." A chest roentgenogram showed no hilar shadows.

The patient was treated with prednisone, 6-mercaptopurine, and radiotherapy to the spleen. She responded well, and eighteen months later she was in good health. The spleen was impalpable, there was no local disease, and the white count was gradually dropping.

Comment.—This case is regarded as one of lymphosarcoma which had entered a leukemic phase. It is interesting because of the unusual site of the primary lesion and the fact that four and one-half years later the patient was alive and well. Initially, the case also was misdiagnosed and a tooth was extracted by the patient's dentist, although the lesion was at first confined to soft tissue.

CASE 3. FOLLICULAR LYMPHOMA-LYMPHOSARCOMA.—The patient, a 42-year-old man who worked as an instrument maker, was first seen at another hospital one year previously (February, 1951). At that time there was a small lump in the substance of the left cheek which was slowly increasing in size. Six months later a small separate lump appeared in front of the left ear. The lump in the cheek (Fig. 5) was excised in December, 1951, and examined histologically. This was reported as follows: "An infiltrating cellular mass of lymphocytes. In some areas there is a follicular pattern, but for the most part this has broken down and it should be regarded as already in a malignant phase" (Dr. George Lumb). At that time there was no evidence of disease elsewhere in the body, and the local lesion was treated by radiotherapy to 2000 r. A small fistula which developed in the cheek was closed by a plastic procedure.

The patient remained well for fifteen months, but in April, 1952, he developed pain in the right hip, right knee, and right shoulder. On x-ray examination, all these sites showed roentgenolucent areas in the bones and were treated by radiotherapy.

A chest roentgenogram, blood count, and sternal marrow examination were all normal. Fifteen months later, in August, 1954, the patient developed generalized lymph node enlargement. In spite of a course of cytotoxic poisons, he followed a downhill course and died four years after the appearance of his first symptom.

Comment.—This patient's initial symptom was a swelling in soft tissue. As the pathologist reported, it had probably entered a malignant phase when the patient was first seen (namely, lymphosarcoma).

Fig. 5.

Fig. 6.

Fig. 5.—Case 3. Follicular lymphoma, site of original lesion. Fistula later closed by plastic procedure.

Fig. 6.—Case 4. Reticulum-cell sarcoma. Swelling of upper lip produced by lesion.

CASE 4. RETICULUM-CELL SARCOMA.—A retired woman of 76 years stated that she had been in indifferent health for about one year and had lost 21 pounds in weight. About six weeks previously she noticed a clear nasal discharge and, at about the same time, a swelling of the upper lip. This gradually increased in size, but there was little associated pain (Fig. 6). Four weeks later a painless lump appeared in the upper part of the left side of the neck.

Examination revealed a rubbery lump in the substance of the upper lip which caused marked external swelling and protrusion. Apart from the lymph node in the left cervical group, there was no evidence of disease elsewhere in the body.

Blood investigation showed hemoglobin 84 per cent and a white blood count of 6,000 per cubic millimeter.

Biopsy of the lip swelling showed a malignant tumor composed of cells with large, pale nuclei, well-marked nucleoli, and moderate cytoplasm. Reticulum stain suggested a diagnosis of reticulum-cell sarcoma (Fig. 7). Complete regression of the swellings followed radiotherapy to the lip and left side of the neck.

After an interval of seven months the patient was readmitted to the hospital with enlarged nodes in the axillae and neck, a palpable liver, a large fixed mass in the pelvis, and a left pleural effusion. Her hemoglobin was 63 per cent, but the white count was normal. In spite of a course of intravenous nitrogen mustard, she went downhill rapidly and died. The total duration of the illness was one year and three months.

Comment.—This tumor in soft tissue pursued a rapid downhill course in spite of an initial response to treatment. Swelling of the upper lip was the presenting sign.

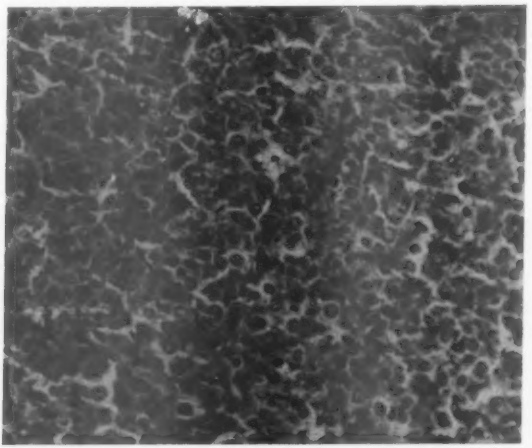


Fig. 7.—Case 4. Histology. Diffuse sheet of reticulum cells suggesting reticulum-cell sarcoma. (Hematoxylin and eosin stain. Magnification, ×200; reduced ⅙.)

CASE 5. RETICULAR LYMPHOMA.—A male storekeeper, aged 46 years, noticed two months previously that his upper lip had become swollen. When he looked at this more carefully, he saw some small swellings in the substance of the lips and the cheeks, under the mucosa. These were painless. He thought that they might have increased in number since first seen. He visited his dentist for advice.

On examination, there were many sagolike swellings under the buccal mucosa of both lips and cheeks. They were about 6 to 7 mm. in diameter, mobile, and painless. The mucous membrane over them had a bluish appearance (Fig. 8).

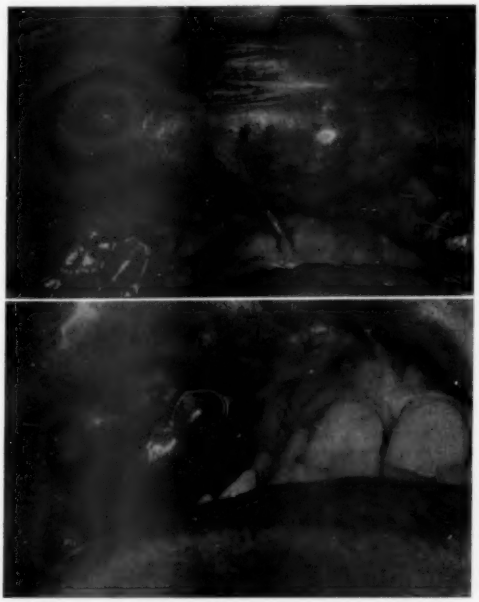
There was diffuse generalized enlargement of lymph nodes up to 5 mm. in the neck and up to 2 cm. in axillae and groins. A chest film and a blood investigation were normal (hemoglobin, 105 per cent; white blood count, 10,800 per cubic millimeter). A nodule excised for biopsy shelled out easily.

The biopsy report was as follows: "A very uniform cellular pattern consisting of mostly lymphocytes with occasional reticulum cells. Mitosis not conspicuous—reticular lymphoma" (Fig. 9). A lymph node biopsy showed similar appearances.

Comment.—The patient was very fit and energetic. Of all the lymphomas, this particular variety carries a relatively favorable prognosis. Radiotherapy to the lips was given for esthetic reasons.

When first seen, the appearances were very bizarre and unusual (namely, multiple submucous swellings). A diagnosis of lymphadenoma was suggested, but the clinical features here almost defy a firm clinical diagnosis.

A.



B.

Fig. 8.—Case 5. Reticular lymphoma. Both *A* and *B* show multiple, bulging submucous swellings.

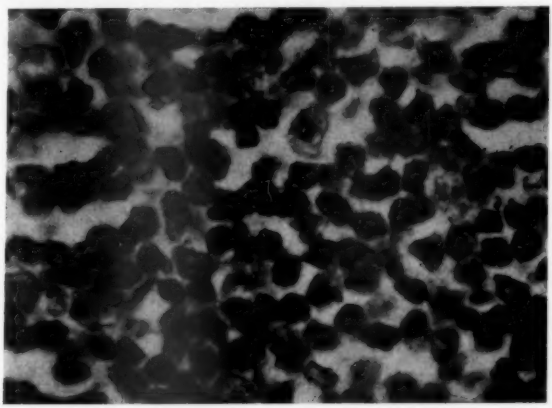


Fig. 9.—Case 5. Histology. Uniform cellular pattern, mainly lymphocytes with occasional reticulum cells. Reticular lymphoma. (Hematoxylin and eosin stain. Magnification, ×1,000; reduced ⅙.)

CASE 6. ANAPLASTIC SARCOMA OF LYMPHOID TISSUE.—A retired 73-year-old man noticed pain in the lower left canine four months previously. The tooth was ground out of occlusion, and this procedure relieved the pain on three separate occasions. After

three months of this conservative treatment the tooth was finally extracted under nitrous oxide anesthesia, as it had become loose. The socket failed to heal, and two weeks after the extraction a swelling related to the socket was seen. The patient had also noticed a tingling sensation in the left lower lip four days after the extraction.

Examination disclosed a swelling of the left lower alveolus extending from the lateral incisor to the second premolar. It was irregular in shape, about 3 cm. in diameter, and of uneven surface without ulceration. It was sessile, painless, firm in consistency, and of maroon color (Fig. 10). There was impaired sensation to a pin prick in the left side of the lower lip.

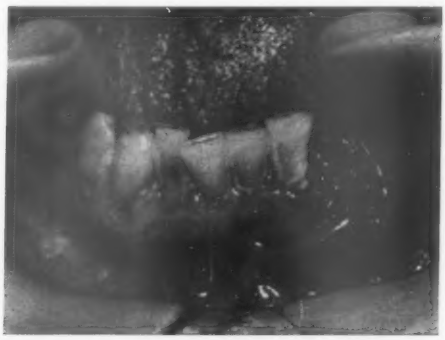


Fig. 10.—Case 6. Anaplastic sarcoma of lymphoid tissue. Note swelling of left lower alveolus involving the area from the lower left lateral incisor to the lower left second premolar.

A. *B.*

Fig. 11.—Case 6. Roentgenogram of left alveolus *(A)* showing diffuse bony destruction in relation to lower left canine. Note soft-tissue outline of swelling. *A*, Sept. 30, 1959. *B*, Oct. 10, 1959.

Enlarged lymph nodes were present in the left and right submandibular groups, and also the left upper deep cervical group. These nodes were smooth, discrete, mobile, and of firm to rubbery consistency.

The nodes in the axillary and inguinal groups were enlarged; the liver edge was just palpable but not hard. The spleen was not felt.

Roentgenographic investigation of the mandible showed a diffuse area of bone destruction, related not only to the apex of the lower left canine but to the whole of the root surface. The outline of this area was irregular and suggested an infiltrating lesion (Fig. 11). A chest radiogram and a blood investigation were normal. Sternal marrow was uninformative. Hemoglobin was 93 per cent, and the white blood count was 7,000

per cubic millimeter. Diathermy biopsy of the swelling showed "a very anaplastic malignant tumor, probably sarcomatous." Biopsy of a lymph node (Fig. 12) was reported as follows: "The lymph node architecture is destroyed and replaced by cellular pleomorphic growth similar to the original biopsy. There are numerous giant cells with convoluted nuclei, and the reticulin is increased. Thought to be anaplastic sarcoma of lymphoid tissue." The patient was given a full course of supravoltage therapy. The oral lesion healed, and the nodes in the neck almost disappeared. However, the patient's general condition deteriorated and he died within two months after being seen.

Autopsy confirmed the healing of the oral lesion and the neck lymph nodes. A part of the small bowel was invaded by growth. The liver was nearly double its normal weight (106 ounces) and filled with small tumor deposits (confirmed by histology). The spleen and para-aortic nodes were also enlarged. Autopsy histology confirmed that the tumor was an anaplastic sarcoma of lymphoid tissue.

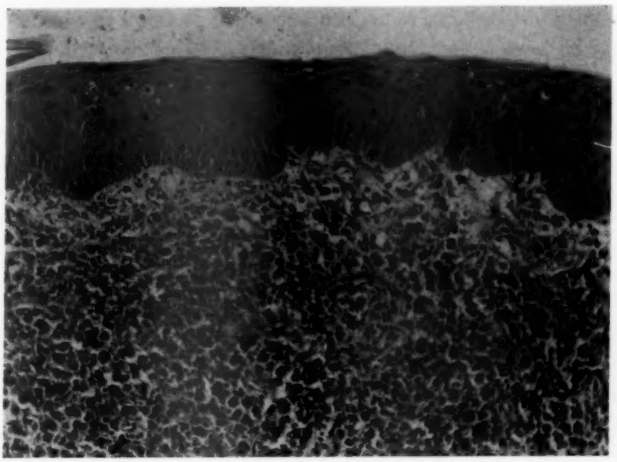


Fig. 12.—Case 6. Histology. Anaplastic cells showing pleomorphism. Many giant cells present. (Hematoxylin and eosin stain. Magnification, ×160; reduced ⅙.)

Comment.—This patient first sought dental treatment for his lesion because of its site. This treatment, in fact, did relieve his pain for a time, but the whole reticuloendothelial system was rapidly involved and death resulted only six months after the first symptoms appeared. It was four months before the correct diagnosis was made, although even early in the illness the roentgenographic appearances were highly suspicious. In some respects, the lesions had features which resembled those of squamous-cell carcinoma.

DISCUSSION

What is the origin of these rare lesions when the first manifest site is in the mouth? Throughout the soft tissues small aggregations of lymphocytes are found scattered here and there, and reticulum cells also occur widely among connective tissues. Drinker and Yoffey[6] have suggested that, in bone, lymphocytes may form in the marrow, derived from cells of primitive mesenchyme. Reticulum cells may be present, interspersed among the endothelial

cells lining blood vessels. If the mature cells are normally present at these sites, then there is no difficulty in explaining tumor origin in them by abnormal cells.

Clinically, lymphomas are frequently (almost invariably) misdiagnosed, a point amply illustrated by this study and by previously reported cases. Because of the site of occurrence, patients usually seek dental advice first (Cases 1, 2, 5, 6 of this series), and this often delays correct diagnosis. Pain in the jaws may be quite unconnected with the teeth, and what is thought to be a simple toothache will not be cured by dental extractions. Superficial lesions which have ulcerated may resemble Vincent's ulcers and may be fruitlessly treated as such until their true nature is revealed. Mistakes may be made even when these cases reach special centers, and early biopsy with diathermy is therefore essential.

It has been clearly shown by the collected evidence that the common presenting sign of these lesions is swelling, with or without accompanying pain. The swelling is usually hard or firm and fixed to the mucous membrane or underlying bone. The site, however, may be one which is not directly contiguous with the teeth, making the possibility of unrelated causes spring more readily to mind. It is in those cases in which the alveolus is directly involved that errors in diagnosis most readily occur.

Another sign that is often present is anesthesia or hyperesthesia of the skin following a regional distribution. This is of the utmost diagnostic importance and must never be ignored, as it commonly indicates infiltration of the nerve trunk by some form of neoplasm.

Therefore, any lesion of unusual appearance with these features is immediately suspect, especially if roentgenographic bone changes of diffuse destruction are present. Immediate biopsy is required for precise diagnosis.

Enlarged regional nodes are often evident and should always be sought. It is quite a striking fact that the oral lesions are often solitary and therefore the patient is first seen at a stage when control of the disease has the best chance of success. Other sites in the body have not as yet been involved, and the response to high-voltage radiotherapy to the local site is remarkably good (Cases 1 and 2).

It is probable that tumors which respond well to treatment do so because of a favorable biologic constitution. Therefore, a histologically very anaplastic lesion does not necessarily carry a poor prognosis. Histologic interpretation of lymphomas is, in any case, a matter for the expert, and even the expert may find it difficult to distinguish between a very anaplastic carcinoma and a lymphoma (Case 6).

Sometimes cases are seen in which generalized gingival enlargement is the presenting sign (Table I, cases of Silverman and Orsós). Biopsy of the gingiva reveals many abnormal lymphocytes, and the white count is increased with an abnormal number of lymphocytes. It is perhaps better to regard these as cases of lymphatic leukemia presenting with lymphadenopathy, rather than lymphosarcoma. The distinction may appear academic, but it can have an important bearing on treatment.

A further point of importance to the dental surgeon is that these cases (Case 2) may present with low platelet counts, thus making postextraction hemorrhage a hazard.

SUMMARY

Six cases of oral lymphoma are described, and their histories and features are given. The literature on this subject is reviewed, and an attempt is made to draw a clearer clinical picture of these rare lesions.

I wish first to thank Sir Stanford Cade for his helpful comments and for permission to include three of his cases in this paper. I am also grateful to Mr. E. S. Lee, Mr. T. M. Prosser, Mr. R. S. Taylor, and Professor A. E. W. Miles for permission to include cases under their care and to Drs. A. D. Morgan and D. H. Mackenzie who kindly produced the photomicrographs.

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DISTANT METASTASIS OF ORAL CARCINOMA

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DISTANT metastasis of oral carcinoma is usually defined as dissemination of the disease to organs or tissues below the level of the clavicles.[4, 10, 13] Early reports of this phenomenon, based upon clinical evaluation without autopsy findings, placed the frequency at about 1 per cent. More recently, however, studies based on postmortem examinations have indicated that oral cancer may spread to distant sites much more frequently.

The present study is based on autopsy findings in 83 of 334 cases of oral cancer seen at Philadelphia General Hospital from Jan. 1, 1945, through Dec. 31, 1954. The autopsy slides were reviewed, and the primary and metastatic lesions were classified according to their degree of histodifferentiation. An attempt was made to determine the influence that age, sex, race, duration of disease symptoms, survival, and histologic structure had on distant metastasis.

REVIEW OF LITERATURE

In 1906 Crile[7] reported that a review of 4,500 cases of head and neck cancer made for him by Hitchings revealed that secondary foci occurred in distant organs and tissues in less than 1 per cent of the cases. Crile concluded that death in cancer of the head and neck almost always results from local and regional development of the disease. Hitchings' findings were evidently based on a review of the literature, and there is no evidence that his study was ever published. However, the concept of the infrequency of distant metastasis of head and neck cancer was popularized by this report, which was amplified in a symposium on oral cancer in 1923.[6, 14] The impenetrability of the collar of lymphatics about the neck was proposed as the reason that metastasis of oral tumors was restricted to regional lymphatic spread.

Ewing[8] observed distant metastases from oral cancer in the liver, heart, adrenal glands, and mesentery, but he stated that such metastases were rare and that extensions to the lungs or pleura were even rarer.

Taylor and Nathanson[15] noted that, except for transitional-cell carcinoma and lymphoepithelioma, oral cancer produces remote metastases only rarely. In their opinion, cancer of the mouth is primarily a local disease, and even in its late stages the sole metastases are usually cervical.

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According to Slaughter,[13] squamous-cell carcinoma has a minimal tendency to spread by way of venous channels but, rather, metastasizes by way of the lymphatics, so that very few metastases below the clavicles are seen. Willis,[20] on the other hand, studying the results of eighty-four autopsies on victims of epidermoid carcinoma of the head and neck, found invasion of the main veins of the head and neck in 50 per cent and distant foci of malignant disease in 41.6 per cent.

Studies supported by autopsy findings have tended to temper the concept of the infrequency of distant metastasis of oral cancer. These studies, with positive findings in 11 to 41 per cent of the cases, are summarized in Table I.

TABLE I. SUMMARY OF LITERATURE ON DISTANT METASTASIS OF HEAD AND NECK CANCER

AUTHOR AND YEAR	PRIMARY SITES INCLUDED	NUMBER OF AUTOPSIES	DISTANT METASTASES	
			NUMBER	PER CENT
Price,[11] 1934	Tongue, lip, floor of mouth, pharynx	87	10	11.4
Burke,[5] 1937	Tongue, lip, antrum, pharynx	28	9	32.1
Braund and Martin,[3] 1941	Head and neck	284	63	23.3
Peltier et al.,[10] 1951	Head and neck	200	34	17.0
Willis,[20] 1952	Head and neck	84	35	41.6
Topazian, 1961	Oral cavity, oropharynx	83	20	24.0

In addition to the studies summarized, Tiecke and Bernier[16] found ninety-one distant metastatic lesions in a review of 401 cases of intraoral carcinoma. The number of patients represented by the ninety-one lesions was not recorded, but all findings were based on postmortem examinations.[2]

Sachs[12] noted distant metastases in four (9.7 per cent) of forty-one autopsies on victims of carcinoma of the tongue.

Martin[9] stated: "If the malignant growth in the mouth and in the neck is not controlled, and if the patient survives long enough, metastasis almost invariably occurs to some viscus below the clavicle, such as the lungs, liver or to the bones."

PHILADELPHIA GENERAL HOSPITAL FINDINGS

Distant metastatic lesions were found in twenty (twenty-four per cent) of the eighty-three cases of oral cancer in which autopsies were performed at

TABLE II. PRIMARY SITES AND DISTANT METASTASES IN EIGHTY-THREE AUTOPSIES

PRIMARY SITE	NUMBER OF AUTOPSIES	DISTANT METASTASES	
		NUMBER	PER CENT
Tongue	26	6	23.0
Tonsil	16	7	43.7
Pharynx	5	0	0.0
Palate	8	2	25.0
Gingiva	2	0	0.0
Buccal mucosa	3	0	0.0
Floor of mouth	16	5	31.2
Lip	7	0	0.0
Totals	83	20	24.0

TABLE III. METASTATIC SITES IN DISTANT SPREAD OF ORAL CANCER

CASE NUMBER	PRIMARY SITE	SITE OF METASTASES								
		HEART	LUNGS	LIVER	SPLEEN	KIDNEY	ADRENAL	DIAPHRAGM	ESOPHAGUS	LYMPH NODES
49390	Floor of mouth						x			
50314	Floor of mouth						x			
51567	Tongue		x							
51748	Tongue								x	Lower esophageal
52263	Tonsil		x							
53313	Palate		x	x						
54497	Tongue	x	x							Substernal
55225	Floor of mouth									Peribronchial
57040	Floor of mouth		x							Periaortic, peripancreatic
57369	Tongue		x							
57714	Tonsil		x		x		x			
58904	Tongue						x			
59114	Floor of mouth	x	x	x	x	x				
59139*	Tonsil	x	x	x		x				Mesenteric
59881	Tonsil		x							Axillary, hilar
60016	Tonsil		x							
62068	Tonsil	x	x	x				x		Mediastinal
62322*	Tonsil		x							
63444†	Palate		x							
3398-J	Tongue		x							

*Transitional cell carcinoma.
†Adenocarcinoma.

Philadelphia General Hospital during the ten-year period from 1945 through 1954. The primary sites involved in these cases are summarized in Table II, and the distribution of the metastatic lesions is presented in Table III.

FACTORS INFLUENCING DEVELOPMENT OF DISTANT METASTASIS

Age.—The ages of nineteen of the patients with distant metastasis were recorded, and the average was found to be 60.5 years. The average age of patients without distant dissemination of disease was 64.2 years.

Sex.—One (five per cent) of the twenty oral cancer patients with distant metastasis was a woman. Of the patients without remote metastasis, 7.9 per cent were women.

Race.—Six (30 per cent) of the patients with distant metastasis and 38 per cent of those without distant metastasis were Negroes. The incidence of Negroes among oral cancer patients at Philadelphia General Hospital during the ten-year period under study was 21.5 per cent, the hospital population being approximately 60 per cent Negro.[17]

Duration of Symptoms.—The duration of symptoms in the patients with distant metastases averaged 5.5 months. Those in whom distant spread was not apparent had an average duration of symptoms of 7.4 months. In many instances, however, the reliability of these data was questionable.

Survival.—The patients with distant metastasis of oral cancer survived an average of 7.9 months from the date of diagnosis, and those in whom remote disease was not apparent survived an average of 8.2 months from the date of diagnosis.

Histopathology.—Among the tumors which produced distant metastases, 35 per cent were differentiated as Grade III and 5 per cent were classified as Grade I. Of the tumors which did not display remote metastasis, 17 per cent were Grade III tumors and 13 per cent were Grade I tumors (Table IV).

TABLE IV. HISTOPATHOLOGIC CHARACTERISTICS AND DISTANT METASTASIS

CLASS	LOCAL DISEASE ONLY	DISTANT METASTASIS	TOTAL
Squamous-cell carcinoma			
Grade I	8	1	9
Grade II	10	2	12
Grade III	12	7	19
Not graded	30	7	37
Adenocarcinoma	2	1	3
Transitional-cell carcinoma	1	2	3

Two of the three transitional-cell carcinomas metastasized below the level of the clavicle, and one of the three adenocarcinomas metastasized to remote regions.

The tumors were not graded when only a single small biopsy specimen was available for study or when the clinical diagnosis was histologically confirmed only at autopsy.

DISCUSSION

The pathogenesis of remote metastasis from cancer of the mouth involves the invasion of the walls of blood vessels. The veins are usually penetrated, since arterial structures show striking immunity to malignant invasion.[1, 21] In support of this observation, Willis[20] has cited the rarity of metastasis peripheral to a primary tumor (regional arterial metastasis).

Emboli, which may consist of tumor cells or tumor-laden thrombi, may be carried to distant sites. The majority of arrested emboli do not develop metastatic lesions,[18, 19] and certain conditions which favor the emigration and growth of emboli after their arrest must be present. Berenblum[1] stated that tissues receiving the emboli must be capable of rapidly providing a blood supply for the new tumor tissue which, in turn, must be capable of withstanding the period before vascularization. He added that only tumor cells at the stem-cell level would be capable of giving rise to metastases, since mature cells would not multiply and proliferate.

There is experimental evidence to support the statement that tumor emboli may pass through such organs as the liver, lung, spleen, and kidney without being arrested.[22, 23]

In this study, fifteen (75 per cent) of the twenty cases in which remote metastases were observed showed involvement of the lungs. In three of the five cases in which the lungs apparently were not involved, distant metastatic lesions were noted only in the adrenal glands.

In all of the cases in which distant metastases were observed there was regional cervical lymph node metastasis.

In one instance (Case 51748) in which carcinoma of the tongue metastasized to the lower end of the esophagus, the metastatic lesion had invaded the deeper layers of the esophagus and left the mucosa and submucosa intact, ruling out the possibility of a second unrelated primary tumor.

In general, the metastases tended to reproduce the histologic structure of the primary tumor, an observation that has been cited by Willis.[20] In one case, the splenic metastasis from carcinoma of the floor of the mouth was of much greater differentiation than the primary lesion.

The belief that distant metastasis invariably occurs in uncontrolled or untreated cancers of the oral cavity was not supported in this study. Fourteen patients admitted to the hospital in a terminal state, having had no previous diagnosis or treatment, survived less than five days after admission. Only three (21 per cent) of these showed evidence of distant metastasis.

It is obvious that factors other than duration and lack of control of the primary lesion should be considered as favoring distant metastasis. The ability of a given tumor to invade vascular structures and the ability of the embolus to remain viable and to proliferate are to be considered.

SUMMARY AND CONCLUSIONS

1. Autopsies were performed at Philadelphia General Hospital on 83 persons who had died of oral or oropharyngeal carcinoma during the ten-year

period from 1945 through 1954. Twenty (24 per cent) of these patients had distant metastasis.

2. Carcinoma of the tonsils, floor of the mouth, palate, and tongue metastasized to remote areas with the greatest frequency.

3. All of the patients with distant metastasis had regional lymph node involvement.

4. The lung was the most common site of distant metastasis, being involved in 75 per cent of the cases.

5. The average age of the patients with distant metastasis was approximately four years less than those in whom distant lesions were not observed. The sex, race, duration of symptoms, and duration of the disease from the date of diagnosis had no apparent effect on development of remote lesions.

6. The majority (35 per cent) of the tumors in this study which produced remote metastatic lesions were Grade III tumors. The incidence of Grade III lesions in the group in which distant metastases were not observed was 17 per cent.

7. Three (21.4 per cent) of the fourteen patients admitted in a terminal state with no previous diagnosis or treatment had distant metastasis. This observation illustrates the fact that factors other than the uncontrolled spread of cancer are operative in the formation of distant metastatic foci of oral cancer.

8. The distant metastasis of oral cancer evidently occurs more frequently than was suspected earlier in this century, and it should be considered in the management of patients with oral cancer, especially when there is regional metastasis.

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THE SO-CALLED ADENOAMELOBLASTOMA

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THE lesion to be discussed in this article is one which, within recent years, has aroused much interest in the literature. Clinically and roentgenographically it may bear strong resemblance to an odontogenic cyst (most often a dentigerous cyst), but histologically it possesses distinctive and unmistakable features. There has been a diversity of opinion concerning the interpretation of these features and the exact nature of the lesion. Thus, it has been referred to by a variety of names, including adenoameloblastoma,[1-3] teratomatous adenoma,[4] pleomorphic adenoma-like tumor,[5] cystic complex composite odontome,[6] and tumor of the enamel organ epithelium.[7]

Stafne[8] first drew attention to the lesion in 1948 when he reported three cases. Since then reports of sixteen additional cases have appeared in the literature.[1, 2, 5-7, 9, 10†] Two more cases are added in this article, bringing the total number of reported cases to twenty-one.

Among these twenty-one cases, except for one in which the patient was a 43-year-old Negro man,[2] the lesions occurred in young persons between the ages of 11 and 26 years. Thirteen occurred in females and eight in males. Distribution between the two jaws was about equal. All but one of the lesions were situated anterior to the region of the first premolars; in the one exception, the lesion occurred near the angle of the mandible.[6] In thirteen cases the lesion was related to an unerupted tooth and resembled a dentigerous cyst, in six cases it resembled a periodontal (lateral) cyst, another case was unique in that the lesion was extraosseous,[9] and there was no record of the relevant features in the remaining case. As to treatment, simple enucleation is consistently reported as having been adequate, and no recurrence has been reported following such treatment.

CASE REPORTS

CASE 1.—The patient, a healthy 12-year-old Chinese boy, was first seen on June 20, 1957. His parents had been concerned over a gradually increasing swelling of the left side of his chin, a deformity which they had first noticed about five months previously. The condition had otherwise been asymptomatic.

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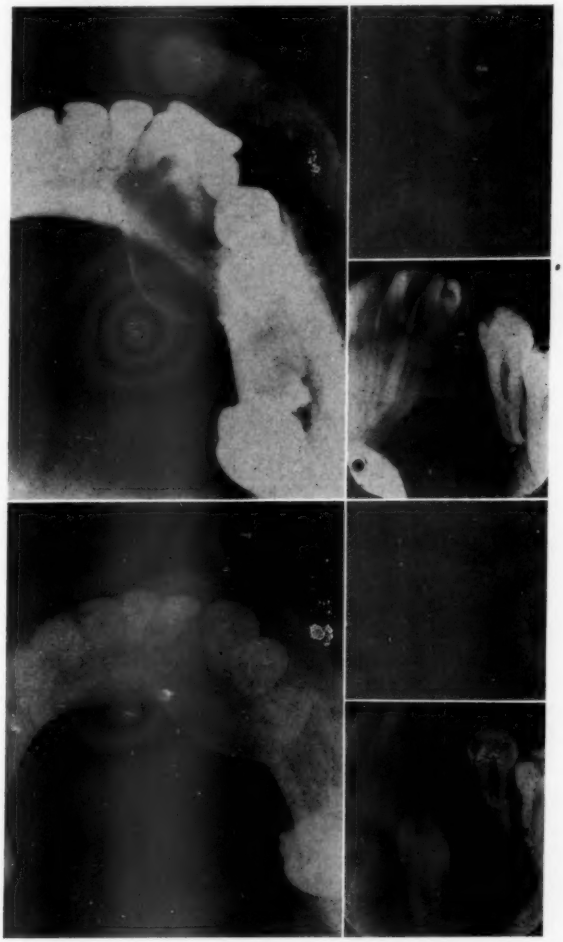
†Thoma's[1] case was duplicated in the series of Bernier and Tiecke[2] (Case 8 in their article), and this has been taken into account. Aisenberg's[3] cases have not been included because of the lack of relevant data.

Examination revealed a well-defined bony expansion of both the buccal and lingual aspects of the mandible, extending from about the midline to the left second premolar region. It "gave" on pressure labially. All the permanent teeth in the involved region had erupted, with the exception of the left permanent canine; the left deciduous canine was still present.

A.



B.

Fig. 1.—Case 1. *A*, Roentgenogram taken when the patient was first seen shows the degree of bony expansion caused by the lesion, which resembles a dentigerous cyst. The involved canine has been displaced inferiorly and a few irregular deposits of calcified tissue may be seen within the cystic space. *B*, Roentgenogram taken five months after marsupialization. The bony expansion has decreased appreciably in size, and new bone deposition has obliterated much of the original cavity. The canine has erupted toward the surface.

Roentgenograms revealed an unerupted left permanent canine (Fig. 1, *A*); associated with it was a large cystic lesion resembling a dentigerous cyst. The involved tooth had been displaced inferiorly, and the lesion had caused distal deflection of the developing roots of the

Fig. 2.

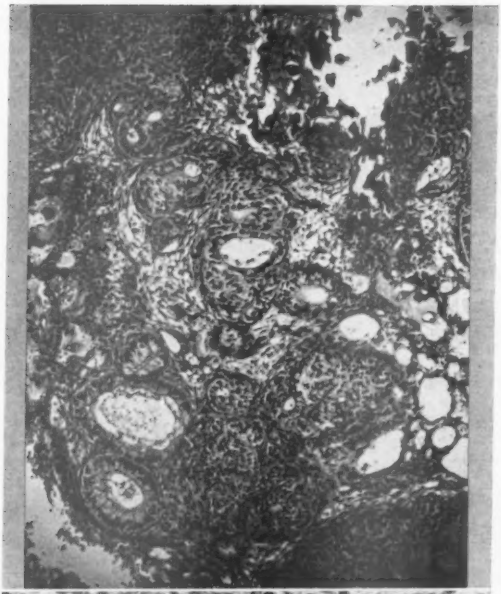


Fig. 3.

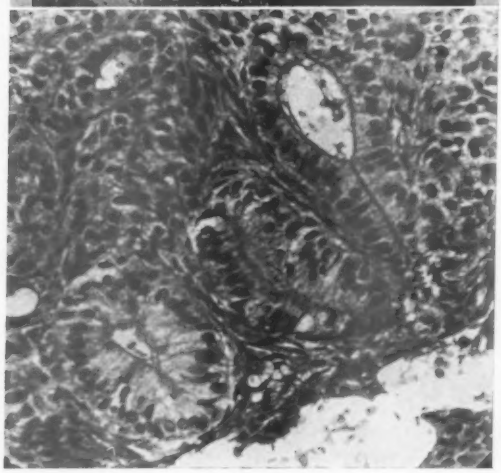


Fig. 2.—Case 1. Tissue removed at first operation showing features typical of the lesion under discussion. Tubule-like structures and other structures consisting of double rows of columnar cells are shown. Fragments of calcified tissue are seen at the upper right-hand corner. (Magnification, ×150; reduced ⅓.)

Fig. 3.—Case 1. Three sausage-shaped bodies are seen. The central body consists of two opposing rows of columnar cells, sandwiched between which is a layer of homogenous material. Within each of the two remaining bodies the opposing rows of cells have become separated at one end by a cystlike space in which the homogenous material appears to have been dragged. (Magnification, ×400; reduced ⅓.)

two left premolars. On close examination of the roentgenograms, small irregular calcified bodies could be detected; they appeared to be situated within the cystic lesion. They were overlooked at the time, however, and the condition was treated as a simple dentigerous cyst.

Treatment.—On June 28, 1957, marsupialization was undertaken under local anesthesia. At operation the cyst lining was found to be unusually thick, but there was, nevertheless, a cystic cavity containing cystic fluid. The left lateral incisor and deciduous canine were extracted, and part of the roof of the cyst adjoining the labial side of the sockets was removed to create a window. The involved permanent canine was left in situ for the purpose of allowing it subsequently to erupt into the arch. It must be emphasized here that during the operation the major part of the cyst lining was left untouched. A temporary gauze pack impregnated with Whitehead's varnish was inserted into the cavity.

Histologic Findings.—The tissue removed at this operation was submitted for routine histologic examination. It was only when the tissue was studied that the true nature of the lesion was realized, for the specimen contained features typical of the lesion under discussion (Fig. 2).

Serial sections (undecalcified) were subsequently prepared. They contained solid clumps of epithelial cells as well as loose cribriform areas[7] consisting of scanty hyalinized connective tissue, blood vessels, and thin strands of epithelial cells.

Conspicuous within the cellular areas were tubule-like structures, each consisting of a central lumen lined by a single layer of tall columnar epithelial cells which, as Miles[6] and Lucas[7] have described, bear a strong resemblance to ameloblasts. These tubule-like structures have been interpreted by other authors to represent ducts.[1-5] However, a study of the serial sections supported Lucas's contention that they were merely cystlike spaces. In none of the sections were there found any structures that could have represented ducts cut longitudinally. Instead, there were several bodies consisting of double rows of tall columnar cells identical to those of the tubule-like structures (Figs. 2 and 3). These bodies appeared sausage-shaped or as short convoluted cords when sectioned longitudinally and as clusters of radially arranged columnar cells when sectioned transversely.

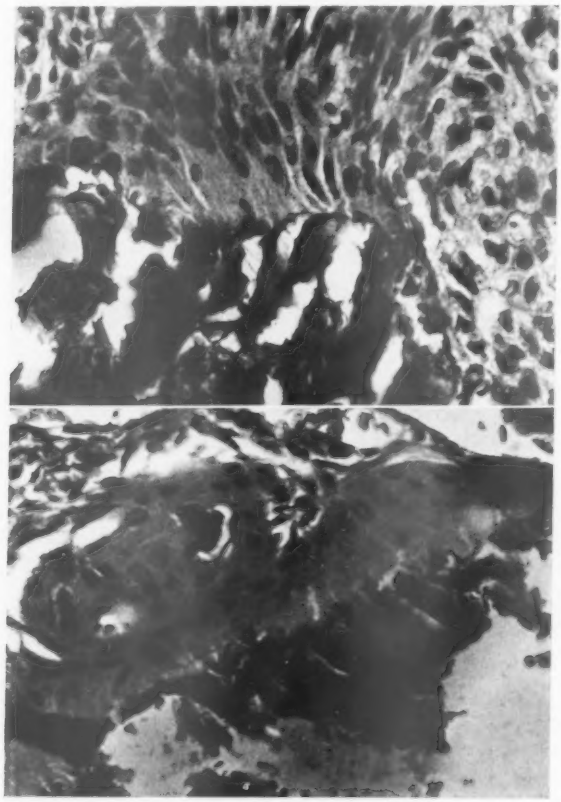
Sandwiched between the two rows of cells, or in the center of a cluster, there was a thin layer of eosinophilic homogenous material. Within some of these bodies the opposing rows of cells had separated in areas and a cystlike space appeared between them (Fig. 3). However, there was still adhering to the inner surfaces of the separated rows of cells homogenous material similar to that which has been described by most authors as occurring in the tubule-like structures. Indeed it appeared as though the smaller of the latter structures represented transverse sections of the sausage-shaped bodies in areas where the opposing rows of cells had become separated. The larger tubule-like structures were probably the result of increase in size of the cystic spaces and separation of the opposing rows of cells along the entire lengths of the original bodies.

The homogenous material has been considered by previous authors to represent preenamel.[4, 6-9] It has been identified by Gorlin and Chaudhry[9] as mucopolysaccharide, which would appear to corroborate this view. Lucas,[7] furthermore, has suggested that the various configurations involving the columnar epithelial cells represent abortive attempts at forming enamel organs. Perhaps this view may be supported by the appearance, in sections of the present case, of areas of loosely arranged stellate cells which bear some resemblance to stellate reticulum (Fig. 2). The stellate cells were continuous with and formed part of "streams" of epithelial cells oriented in such a way as if to form an epithelial "stroma" to the well-defined sausage-shaped and tubule-like structures; in many areas these cells were compressed and were spindle-shaped, but in the looser areas they assumed stellate forms.

Several calcified bodies were also found scattered throughout the sections. There were small ovoid bodies as well as larger masses which had become fragmented as the result of preparation. They gave a positive reaction for calcium with von Kossa's stain, assumed a deep blue with Mallory's triple stain, and stained yellow with van Gieson's stain. An interesting and significant finding was the presence of rows of tall columnar epithelial cells in direct contiguity with some of the fragments of the larger calcified deposits; they ap-

peared as though engaged in the production of the latter (Fig. 4, *A*). These cells resembled ameloblasts even more than did the columnar cells found elsewhere in the sections; in general, they were taller and their cytoplasm was more uniformly and deeply stained. (The cytoplasm of other columnar epithelial cells was lightly stained and had a reticular appearance.) In common with the other columnar cells and with ameloblasts, the nuclei of these cells were uniformly arranged at the poles opposite the calcified deposits. Columnar cells of this nature have not hitherto been reported in the lesion under discussion. They were clearly in evidence in the second case reported in this article and were also detected when sections from my previous case[5] were examined for them (Fig. 4, *B*).

A.



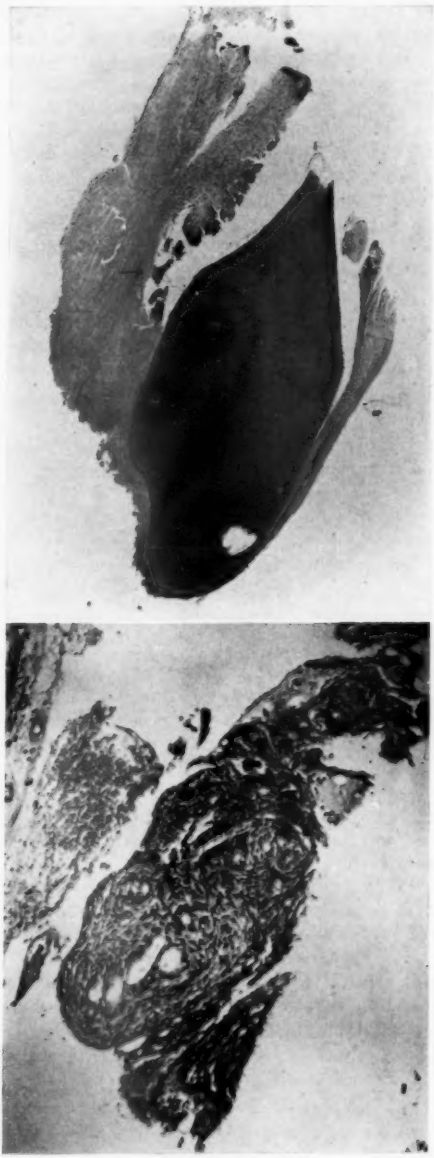
B.

Fig. 4.—Tall ameloblast-like cells are shown in direct contiguity with fragments of calcified tissue. *A*, From Case 1; *B*, from previously reported case.[5] (Magnification, ×600; reduced ⅓.)

Postoperative Progress and Treatment.—It was decided deliberately to withhold further surgery and to study the postoperative behavior of the lesion.

After a few weeks it became obvious that the lesion was regressing and the involved canine was erupting toward the surface. Roentgenograms showed radial deposits of new bone against the original wall of the cavity, and there was a decrease in the bulge of the alveolar bone.

A.



B.

Fig. 5.—Case 1. Sections of specimen removed at final operation. *A*, The crown of the embedded tooth is surrounded, except at its incisolabial aspect, by a mass of soft tissue consisting essentially of collagen fibers lined internally by stratified squamous epithelium. *B*, High-power view of area marked by arrow in *A*, in which features of the original lesion are seen. (Magnifications: *A*, ×65; *B*, ×150; both reduced ⅕.)

Later it was noted that the canine's rate of eruption lagged behind the rate of obliteration of the cyst cavity; the crown of the canine became partially submerged in soft tissue which, clinically, appeared to be normal tissue.

On Aug. 12, 1957, under local anesthesia, a portion of this tissue was removed and the crown of the tooth was uncovered.

Histologic examination of the excised tissue showed it to contain a thick layer of collagen fibers, scattered within which were a few epithelial cell rests. There was a lining of rather hyperplastic stratified squamous epithelium. Immediately below the epithelial lining was granulation tissue, which is normally seen in a receding cyst. The sections contained none of the typical features of the original lesion.

It was later noted that the position of the canine became static and its crown again became partially covered by soft tissue. By this time the lesion had grown appreciably smaller in comparison with its original size (Fig. 1, *B*).

On Dec. 12, 1957, under local anesthesia, the lesion was finally removed together with the involved tooth. It shelled out readily. The bony cavity left behind was somewhat rough due to new bone deposition. Postoperative progress was uneventful. To date there has been no evidence of recurrence.

Histologic Findings.—The specimen consisted of a pear-shaped mass of soft tissue, largely fibrous tissue, surrounding all but the incisolabial part of the crown of the canine.

Decalcified sections of the specimen showed a thick layer of collagen fibers surrounding all but the incisolabial portion of the crown (Fig. 5, *A*). It was lined internally, for the greater part by stratified squamous epithelium. Epithelial cells with the characteristics of the original lesion were found to be present only opposite the cervicolingual region of the crown of the embedded tooth. Within a small concentrated area a few tubule-like structures were seen, together with a few calcified bodies (Fig. 5, *B*).

CASE 2.—A Chinese man, aged 20 years, was seen on Oct. 20, 1958, for treatment of a swelling of his upper lip. He had first noticed the swelling about one year previously, and it had gradually increased in size since then.

On examination, a large cystic lesion was found to involve the maxilla. This extended from the region of the right first premolar to a little left of the midline. It had caused a pronounced labial expansion of the involved bone with resultant displacement of the right side of the upper lip, the right ala of the nose, and the adjoining tissues of the face. There was also a fullness of the palate in the involved region. Fluctuation was detected labially.

A metal bridge extended between the two lateral incisors with a pontic replacing the right central incisor, the only tooth found to be missing from the arch. The patient could not recall having had this tooth extracted previously.

Roentgenograms showed the right central incisor to be unerupted and associated with what at first appeared to be a dentigerous cyst (Fig. 6). A few irregular calcified bodies were seen superimposed on the cystic space, however, and they aroused the suspicion that the lesion was likely to be similar to the one under discussion.

On Oct. 31, 1958, about 2 c.c. of straw-colored fluid was aspirated. The swelling partially collapsed after this, but by the following day it had regained its original size.

A biochemical examination of the aspirated fluid produced the following findings:

Total proteins	4.9 Gm. per cent
Albumin	2.7 Gm. per cent
Globulin	2.2 Gm. per cent
Cholesterol	410.0 mg. per cent
Potassium	15.3 mg. per cent
Sodium	340.0 mg. per cent
Mucin	"Present"

Treatment.—The patient was admitted to the hospital on Dec. 2, 1958. On the following day the lesion was enucleated under general anesthesia. A labial flap was reflected and

thin bone, found to be incompletely overlying the labial aspect of the lesion, was removed with rongeur forceps. The lesion, which possessed a thick fibrous capsule, was readily shelled out in toto, leaving a smooth-walled cavity in the maxilla. Unfortunately, the involved central incisor became separated from the lesion in the process. A portion of the root of the right lateral incisor was found to be denuded, and the tooth was extracted. A temporary gauze pack impregnated with Whitehead's varnish was inserted into the cavity.

Healing progressed uneventfully, and when the patient was last seen on Aug. 25, 1959, a shallow labial depression was all that remained of the original cavity. There was no evidence of recurrence.

Fig. 6.

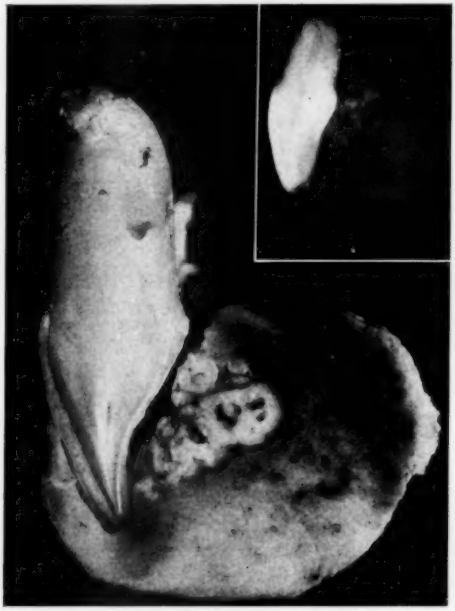


Fig. 7.

Fig. 6.—Case 2. The lesion resembles a dentigerous cyst involving the unerupted right central incisor. A few irregular calcified bodies are seen within the cystic cavity.

Fig. 7.—Case 2. Cut surface of specimen with the embedded central incisor. (Magnification ×5.2; reduced ⅙.) *Inset:* Roentgenogram of specimen showing deposits of calcified tissue within the lesion.

Specimen.—The specimen was roughly spherical in shape and measured approximately 2.25 cm. in diameter. The crown of the central incisor fitted exactly into a depression on its surface. Although clinically the condition was undoubtedly cystic in nature, the specimen itself appeared as a solid mass of fibrous consistency. Roentgenograms of the specimen displayed irregular calcified bodies within the soft tissue.

The specimen was cut axially, together with the embedded tooth (Fig. 7). The cut surfaces of the lesion also gave the appearance of a solid mass consisting of a thick fibrous capsule. Against the labial surface of the embedded tooth there was a collection of grayish white tissue; over this area the surface felt gritty.

Histologic Findings.—One-half of the cut specimen was decalcified, together with the embedded tooth, and serial sections were prepared. Undecalcified sections were also prepared from the second half of the specimen.

The sections consisted of a thick connective-tissue capsule lined internally by a thin layer of stratified squamous epithelium resembling cystic epithelium. This epithelial lining was replaced opposite the lingual surface of the crown of the embedded tooth by an epithelial tissue mass which appeared to obliterate much of the collapsed cystic space.

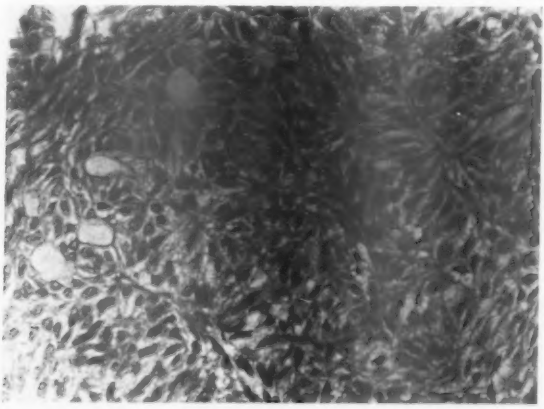


Fig. 8.—Case 2. In this field, instead of well-developed tubule-like and sausage-shaped structures such as those seen in Case 1 and in other reported cases, there are what appear to be the same structures in an immature stage of development. (Magnification, ×400; reduced ⅓.)

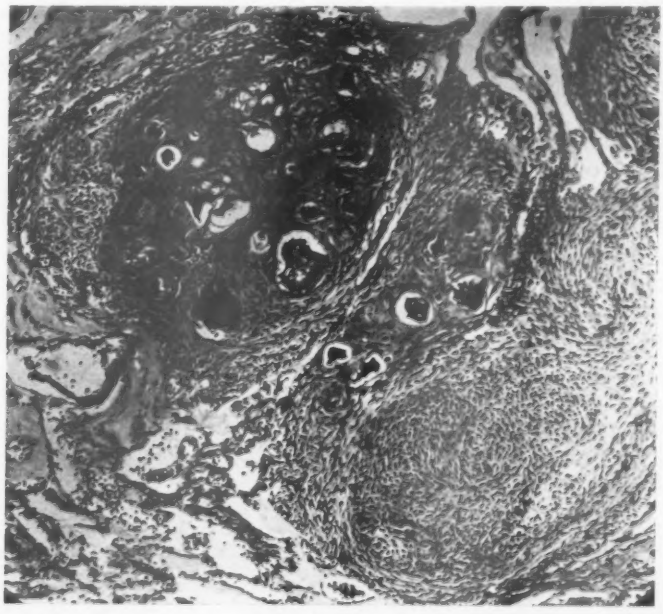


Fig. 9.—Case 2. Decalcified section showing the organic remnant of deposits of "enameloid tissue" surounded by their "formative" cells which are more compact and densely stained than the main cellular elements of the lesion. An example of the latter is seen below and to the right; it contains no tubule-like or other allied structures. (Magnification, ×150; reduced ⅓.)

The epithelial mass bore the same general features as the lesion under discussion, although the well-developed tubule-like and sausage-shaped structures which formed outstanding features in other cases were absent (Fig. 9). There were, however, solid cellular clumps interspersed with loose cribriform areas containing blood vessels and thin strands of epithelial cells in a stroma of hyalinized connective tissue.

The solid cellular clumps consisted of young active-looking epithelial cells; there were spindle-shaped epithelial cells oriented around collections of other plump epithelial cells. Among some of the latter cell groups could be seen clusters of cuboidal pale-staining cells arranged radially around thin traces of eosinophilic homogenous material similar to that described in other cases. These clusters of cells appeared to represent immature sausage-shaped structures; they were ill-defined but, nevertheless, recognizable. In their vicinity could be seen a few small, also seemingly immature, tubule-like structures lined with cuboidal cells (Fig. 8).

In other areas there were conspicuous clumps of deeply stained epithelial cells which contrasted with the surrounding pale-staining cells. Calcified masses were present within these clumps (Figs. 9 and 10, *A*). They ranged from small ovoid bodies to larger irregular masses; they had the same staining reactions as enamel to van Gieson's stain, hematoxylin and eosin, Mallory's triple stain, and silver stain, and in undecalcified sections they produced a positive reaction for calcium with von Kossa's stain. In many respects, the ovoid bodies resembled the "enameloid globules" which Mezl[11] has described as arising in relation to the development of simple enamelomas; as they expanded, they fused with one another to form larger irregular bodies in exactly the same way as described by Mezl. In some there were also deposits of a calcified structure with a lamellar appearance similar to Mezl's "enameloid lamellae." Much of the "enameloid tissue" appeared to have undergone maturation judging by the loss of structure in decalcified sections. In addition to these bodies, there were irregular calcified masses in direct contiguity with which were rows of columnar ameloblast-like cells similar to those described in Case 1. Their relationship with the calcified tissue was well shown in the decalcified sections, although only the organic matrix of the latter tissue was left (Fig. 10, *A* and *B*). No prismatic bodies were detected in these calcified masses.

A few of the larger calcified masses appeared to be lying loose within connective tissue stroma, but they were usually surrounded by inactive squamous epithelial cells which were also found to occupy spaces within the masses. These cells presumably represented remnants of the original formative cells following cessation of activity. No calcified tissue of mesodermal origin could be detected in any part of the lesion.

DISCUSSION

There can be little argument against the contention that the epithelial components of the lesion under discussion are derived from the epithelial cells of the enamel organ or its remnants, as is held by Lucas[7] and others. It is possible that the lesion may arise directly as the result of aberrant proliferation of these cells, as in the case of the "solid" lesions reported. It is also possible, however, that it may arise indirectly from the same cells in the sense that it may originate from the epithelial lining of an odontogenic cyst. The lesions that resemble dentigerous cysts lend support to this view. In the second case reported here the histologic characteristics were such as to suggest that the lesion was in an immature stage of development; yet, in this 20-year-old man, the involved central incisor had remained unerupted and was associated with a relatively large cystic lesion from which typical cystic fluid was aspirated.

The manner in which the proliferating cells differentiate has been a subject of divergent viewpoints. The adenoid appearance of the lesion on histologic

A.

B.

Fig. 10.—Case 2 (decalcified). *A*, In this field a portion of the surrounding connective tissue capsule is shown (top left), together with some of the epithelial cellular elements of the lesion. Near the center there are organic remnants of "enameloid tissue." A row of tall ameloblast-like cells may be seen to surround a large deposit (arrow). *B*, A high-power view of the ameloblast-like cells. (Magnifications: *A*, ×150; *B*, ×720; both reduced 1/5.)

examination has led some authors to believe that there has been an attempt to form glandular tissue.[1-5] They have argued that the cells involved in the lesion, having originally been derived from the basal cells of the oral epithelium, retain the potentiality of the latter cells to differentiate into either odontogenic or glandular epithelium; it would not seem unreasonable to suggest that under unusual conditions (such as those which obtain during the growth of the lesion under discussion) they may differentiate in either direction.

In so far as the present lesion is concerned, however, in the light of data of the various cases reported and on further study of my own cases, the evidence rather supports the view that differentiation has occurred essentially toward an attempt to form enamel rather than glandular tissue. The acini of the tubule-like structures which constitute an outstanding feature of the lesion and which contribute largely, if not solely, to its adenoid appearance have been shown by Lucas[7] merely to represent cystic spaces. A detailed study of serial sections of my own cases supports this view. The noncalcified eosinophilic homogenous material and the tall columnar cells associated with these tubule-like and other allied structures have been demonstrated by Lucas,[7] Gorlin and Chaudhry,[9] and others to represent an abortive attempt at enamel formation. A further and perhaps more significant result of this attempt is the consistency with which calcified bodies have been detected in all cases reported. In some cases there were heavy deposits, while in others the deposits were scanty and widely distributed. The calcified bodies frequently appeared as ovoid deposits which often fused together to form larger irregular masses; the appearance of these bodies, their relationship to the "formative" epithelial cells, and their staining characteristics closely resembled "enameloid tissue" which has been described by Mezl[11] as occurring in relation to the development of simple enamelomas as an attempt of the lowly differentiated epithelial cells concerned to form enamel in the absence of any "organizing and formation-inducing functions of the pulp." The appearance of rows of ameloblast-like cells in direct contiguity with other calcified deposits which also produced the same staining reactions as enamel, as described in both of the present cases, provides further important evidence of attempted enamel formation. This indicates an advanced degree of differentiation, though also without any apparent mesodermal influence which otherwise would probably have induced full differentiation into ameloblasts and subsequent formation of dentin as well as true enamel, as in the case of a complex composite odontome. Although Miles[6] reported the presence of calcified tissue resembling dentine in the case that he saw, I was unable to detect any such tissue in my own cases. Mezl has pointed out that odontogenic epitheliums "may have a certain independence in exerting some of their functions," as has been borne out by various experiments and observations. This may explain the production of calcified enamel-like tissue in the absence of dentine in the lesion under discussion.

The question of whether the lesion itself is neoplastic in nature is also open to debate. Certainly, the lesion behaves in a manner significantly different from the ameloblastoma, of which it is regarded by some authors[1-3] to be a

variant form. It does not have the locally invasive characteristics of an ameloblastoma; nor does it show the slightest tendency to recur following simple enucleation. There are also significant histologic differences between the two conditions.[6, 7] It is therefore unfortunate that the lesion has been linked with the ameloblastoma.

Lucas maintains that the lesion, though not an ameloblastoma, is a true neoplasm of the enamel organ since it displays "persistence of growth." Against this view can be placed the behavior of two of my own cases following simple marsupialization (Case 1 of this article and a case reported previously[5]). In both cases there was ample evidence that "tumor tissue" had been left in situ following the operation; yet, in both cases there was appreciable regression in size of the lesions and the involved teeth were able to erupt further toward the surface. Furthermore, the seeming disappearance of "tumor tissue" in final sections of both cases and the appearance in one case of inactive squamous cells in association with calcified deposits gave the impression that the lesion had, to a large extent, "burned out." Therefore, any increase in the size of an untreated lesion is more likely to be due to simple cystic expansion of either the cyst from whose wall the lesion developed or that developed as the result of cystic degeneration within a "solid" lesion (as described by Lucas).

Both Miles[6] and Cahn[4] place the lesion in the category of a developmental abnormality. Miles compares it with the complex composite odontome, but his suggestion that there had been mesodermal participation in the development of the lesion in his case has not been borne out in other cases. Cahn, however, compares the lesion with a teratoma in view of his contention that there had been dual differentiation into both odontogenic and glandular epithelium and also in view of the formation of "cementicles," which he interpreted the calcified deposits to be. The evidence presented in this article also favors classification of the lesion as a self-limiting developmental abnormality involving the enamel organ epithelium or its remnants, though not of either of the forms suggested by Miles and Cahn.

SUMMARY

Two more cases of a lesion often referred to as "adenoameloblastoma" are reported.

This lesion is believed to be a form of developmental abnormality derived, directly or indirectly, from epithelial cells of the enamel organ or its remnants and involving an attempt at enamel formation.

Emphasis is placed on the consistency with which calcified deposits have been detected in all cases so far reported. In the present cases the calcified deposits are shown to resemble enamel or "enameloid tissue," both in staining characteristics and in their relationship to "formative" epithelial cells. Some of the latter cells show strong ameloblast-like characteristics.

My thanks are due K. P. Yap who prepared the photographs and microphotographs used as illustrations and K. K. Chee and Simon Foo who prepared the sections.

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AMELOBLASTIC ODONTOMA

Report of a Case

A. Forsberg, L.D.S., M.D., C. Lagergren, M.D., and G. Mårtensson, L.D.S., M.D., Stockholm, Sweden

THE classification and nomenclature of odontomas leaves much to be desired as regards consistency. One acceptable scheme would seem to be that published by Pindborg,[3] who defines the odontoma as an odontogenic tumor that has attained a degree of tissular differentiation in which dentine, enamel, and often also cementum have developed. Pindborg divides odontomas into three main groups: amelobastic, complex, and composite. The ameloblastic odontoma does not constitute an independent group, however, but may be regarded as an intermediate stage in the development of the odontoma. The earliest stage in this chain of development is a genuine soft-tissue tumor, which has been designated an ameloblastic fibroma or soft odontoma, and the mature product is a completely mineralized odontoma of the complex or the composite type.

Synonyms for ameloblastic odontoma are adamanto-odontoma, soft and calcified odontoma,[2] and odontoameloblastoma.[4] In Thoma's[5] often-quoted scheme the ameloblastic odontoma seems to be included in the complex odontoma group.

The ameloblastic odontoma presents a varied histologic picture. It contains hard-tissue substances (namely, dentine, enamel, and sometimes cementum), accumulations of ameloblast-like epithelial cells arranged in the form of islands or strings, and connective tissue which is usually embryonal in type.

From the number of cases published, it would seem that the ameloblastic odontoma is seldom encountered and is the least common of the three groups.[1] This is consistent with our personal experience.

The clinical symptoms of odontoma are usually insignificant, and the diagnosis is generally made on routine examination of the dentition. Often a tooth is missing, with no history of extraction. The odontoma grows slowly and can gradually cause local swelling in the jaw. Other symptoms, such as neuralgic pains due to compression of the nerve, are less common. Odontomas may sometimes become infected, and the process may consequently be diagnosed as osteitis in the jaw with sequestra.

CASE REPORT

The patient was a 10-year-old girl. Routine examination at the school dental clinic revealed a swelling at the site of the lower left first molar, which was missing. The patient was admitted to the Caroline Hospital for further examination.

From the Ear, Nose and Throat Clinic (Head: Prof. T. Skoog) and the Roentgendiagnostic Department (Head: Prof. K. Lindblom), Karolinska Sjukhuset, Stockholm 60, Sweden.

Roentgenographic examinations (Figs. 1 and 2) showed, at the site of the missing tooth, a roentgenolucent area about 1 cm. in diameter, surrounded by a narrow sclerotic border. In this region the mandible was slightly swollen and the cortical layer was thinned, both orally and vestibularly. The roentgenolucent area continued distally as a process passing over the crowns of the still unerupted second and third molars. In the roentgenolucent region there were small areas of a granular mineralization. A deciduous tooth was situated in contact with the anterior margin of the translucent area, and its distal root had been resorbed. A provisional diagnosis of immature odontoma was made.

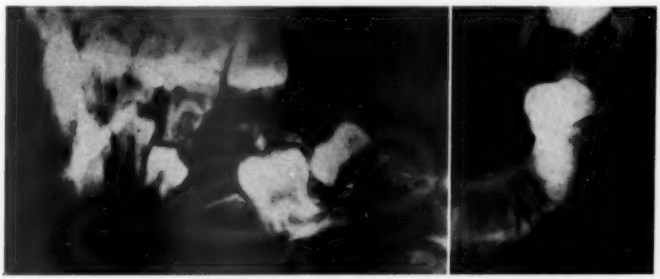

Fig. 1. Fig. 2.

Figs. 1 and 2.—Roentgenograms of an ameloblastic odontoma situated in the left part of the mandible.

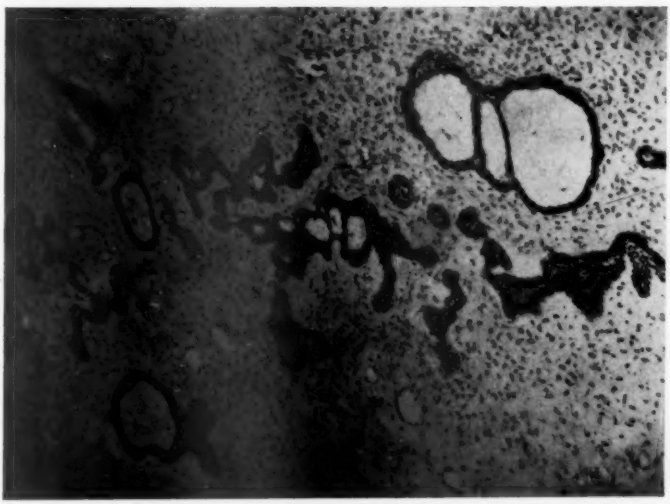

Fig. 3.—Photomicrograph of ameloblastic odontoma. The tissue in this area consists of epithelial cells and connective tissue of embryonal type. (Hematoxylin and eosin stain. Magnification, ×216; reduced ⅔.)

Operation.—The tumor was extirpated under intubation narcosis. A section through the gingiva and the periosteum was made in the vestibulum from the second left premolar to the third molar. The lateral part of the mandible was exposed by dissection. The marginal part of the region showed partial destruction by a soft, grayish tumor. Small areas of mineralization were found in the tumor tissue. The tumor was radically excavated, and primary suturing was done. The recovery was uneventful, and the wound healed satisfactorily.

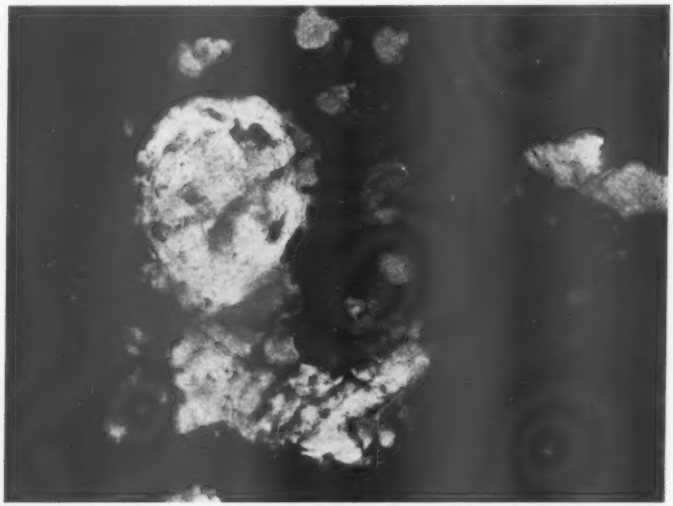

Fig. 4.—Ameloblastic odontoma. Microroentgenogram. (Magnification, ×150; reduced ⅔.)

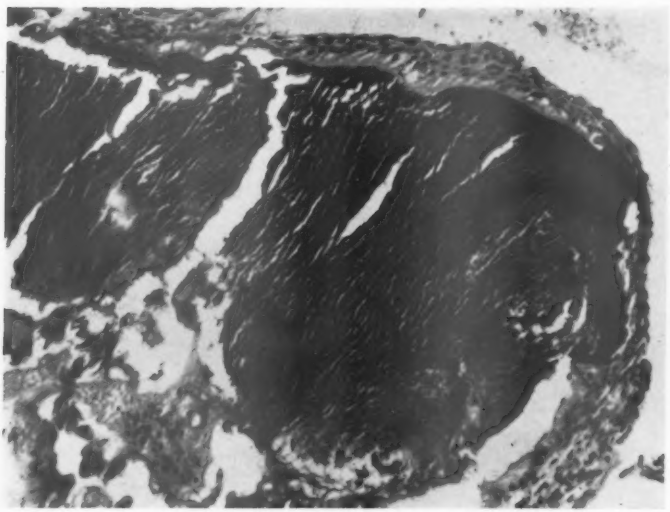

Fig. 5.—Ameloblastic odontoma, decalcified specimen, containing immature enamel. On part of the enamel surface there are ameloblasts. (Van Gieson's stain. Magnification, ×216; reduced ⅔.)

Histologic examination of the tumor showed that it was composed of connective tissue of the embryonal type, containing small groups and strings of epithelial cells (Fig. 3). In the periphery of the epithelial accumulations there were, in many places, palisade formations of cylindrical epithelial cells. Centrally in the groups some of the cells were of the stellate type, and a transition to small epithelium-covered cysts was observed. There was no appreciable cell polymorphism. On the surface of the tumor, connective tissue (rich in collagen fibers) and some bone fragments were seen; the latter were in places diffusely demarcated from the tumor itself. The bone fragments in some sections were eroded by the tumor tissue. There was no distinct encapsulation. In the tumor mass there were small mineralized areas in which microroentgenographic examination revealed no definite structure (Fig. 4). In the decalcified specimen these were found to consist of immature enamel, the organic substance of which did not decompose on decalcification (Fig. 5). On part of the enamel surface there were ameloblasts. At other places there was a small mesenchymal covering which stained red with van Gieson's stain. This might have represented an early stage of dentine matrix; however, since no dentinal tubules could be seen, it could not be classified with certainty as dentine.

DISCUSSION

Differential diagnosis in the foregoing case presented no major problems. The case history and clinical findings excluded the possibility of osteitis. From the roentgenographic findings, it was fairly certain that the formation consisted of an odontogenic tumor. The small roentgenopaque granules in the roentgenolucent area suggested that this was not a real soft-tissue tumor, such as an adamantinoma. Of the partly mineralized tumors, the choice lay between ameloblastic odontoma, soft odontoma with epithelial mineralization,[6] and possibly dentinoma. The differential diagnosis between these tumors, which is of no real practical significance, had to be based on histologic examination.

SUMMARY

An ameloblastic odontoma in a 10-year-old girl was detected at a routine school dental examination. The diagnosis was made on the basis of roentgenographic and histologic studies.

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ENDODONTICS

The American Association of Endodontists

Captain Warren J. Hedman, Editor

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REACTION OF HAMSTER TISSUE TO DRUGS USED IN STERILIZATION OF THE ROOT CANAL*

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INTRODUCTION

THE use of chemotherapeutic agents for the treatment of dental disease has increased in popularity in the past fifty years. This is especially true in the field of endodontics, where the adoption of such agents as a means of achieving asepsis of the infected root canal has assumed significant importance. In view of the fact that any medicament used in this capacity ultimately contacts viable tissue, it is desirable that its reaction with this tissue be in no way deleterious to the inherent physical and chemical life processes of the cells of which the tissue is composed. In instances where these drugs are not compatible with cellular activity, success of the treatment is often delayed and sometimes not realized due to the reduced regenerating power of the area subjected to chemical injury.

Unfortunately, not all of the root canal antiseptics presently employed in endodontics are tissue tolerable. A study was therefore undertaken to analyze the irritating potential of these agents and to determine their suitability for use in the root canal.

REVIEW OF THE LITERATURE

A review of the literature reveals that several investigations have been undertaken in an attempt to study the irritating potential of root canal medicaments. The earliest reported investigation, presented by Peck[25] in 1898, consisted of an evaluation of the compatibility of various root canal drugs with

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skin tissue. This was accomplished by applying a cotton pellet saturated with a test solution to the skin of the forearm with a rubber cup and adhesive tape for a period of twenty-four to forty-eight hours. The irritability of the drug was evaluated by the degree of inflammation, blistering, discoloration, and tenderness produced. In addition, Peck also tested the effect of a spray of these drugs on the rate of healing of wounds mechanically induced on the abdomens of guinea pigs. Using these methods, Peck tested the effect of oil of cassia, oil of peppermint, oil of cloves, oil of cinnamon, oil of bay, oil of cajeput, oil of myrtle, oil of sassafras, beechwood creosote, 95 per cent carbolic acid (phenol), Black's 1, 2, 3, solution (1 part oil of cassia, 2 parts phenol, 3 parts oil of gaultheria), eucalyptol, and formalin. He found that phenol, formalin, and oil of cassia all produced severe tissue damage and induced a marked delay in wound healing. Eucalyptol and oil of cloves produced no tissue irritation and increased the rate of healing. The remaining drugs, he reported, produced little or no irritation and had no significant effect upon the rapidity with which the wounds healed.

The forearm test introduced by Peck was also used in subsequent investigations by Black[3] in 1920, Grossman[13] in 1944, and Buchbinder[4] in 1947. Black,[3] who repeated several of the agents tested by Peck, observed a more severe reaction to oil of cassia and oil of cinnamon. He attributed this to differences in the purities of the oils tested in the two experiments. Black also included formocresol, a drug not used by Peck, in his studies. He found this agent to be grossly irritating, comparable in severity to both formalin and phenol.

Grossman,[13] who conducted his forearm experiments more than twenty years later, tested only those drugs employed in endodontics at that time. These included Azochloramid in triacetin, camphorated parachlorphenol, Cresatin, formocresol, and beechwood creosote. Formocresol, he reported, produced a severe tissue necrosis similar to that found by Black. Unlike both Black and Peck, however, Grossman observed that beechwood creosote, though producing no necrosis, did induce a severe inflammation which persisted for approximately seven days. No irritating effects were noted when the three other drugs were tested.

Buchbinder[4] slightly modified the forearm technique used earlier by the other researchers, by mechanically producing a scratch wound on the skin prior to application of the test drug. He then tested two drugs (penicillin and eugenol) over a twenty-four-hour period. Upon removal and inspection of these drugs, he reported that neither site on the subject's arm showed any sign of irritation or delayed healing.

An investigation which later became a source of reference for several articles dealing with the irritability of root canal medicaments was reported by Coolidge[5] in 1932. In his report, Coolidge described the microscopic reaction of the periapical tissue of pulpless teeth in dogs to drugs which were sealed in the root canal for twenty-one days. The drugs—oil of cloves, eugenol, eucalyptol, phenol, Cresatin, hexylresorcinol, formalin, formocresol, 4 per cent chloramine, and chlorinated soda—were compared with $\frac{1}{100}$ normal sodium hydroxide which was used as a control and found to produce only a very mild

reaction at the apex. Eugenol and eucalyptol, on the other hand, were found to produce extensive leukocytic infiltrations and osteoclasis. In tissue which had been exposed to phenol, Coolidge described moderate inflammation with coagulation of protein and necrosis of tissue surrounded by a connective-tissue capsule. Teeth treated with formocresol showed a less extensive area of necrosis, with only a limited amount of cellular infiltration. Cresatin, hexylresorcinol solution, and 4 per cent chloramine all produced extensive leukocytic accumulations, with the latter causing an initiation of tissue breakdown but no protein precipitation. Formalin produced considerable inflammation and tissue fixation. No irritation of periapical tissue was caused by either oil of cloves or chlorinated soda.

A monograph dealing with the subject of chemical periodontitis in dogs was also published by Feldmann[8] in 1930 and discussed later by Kronfeld.[17] It was Feldmann's opinion that all drugs sealed in root canals at first caused an inflammatory infiltration of the periapical tissues. After a period of several months, he found, the condition of the periodontal tissue varied according to the kinds of drugs used and the length of treatment. Roots that had been filled with gutta-percha or oxyphosphate cements following the use of mild drugs were surrounded by normal periodontium. In those in which harsher, more penetrating drugs, such as 40 per cent formaldehyde and tricresol formalin, were used, there appeared progressive inflammation with destruction of apical hard and soft tissues. No tissue repair was evident.

Chemotrauma produced by drugs used in root canal therapy was also studied by Robinson and Koch.[29] The investigative technique used by these researchers consisted of first exposing the pulps of cat's teeth and then allowing them to be contaminated by saliva for various periods of time. Some animals were given an additional parenteral injection of a 1 c.c. suspension of staphylococcus and streptococcus. The teeth were then treated with penicillin, sulfathiazole, beechwood creosote, tricresol, pyridyl mercuric chloride, and propyl mercuric chloride. Teeth left untreated were used as a control. After varying periods of time, the jaws were removed and radiographic, histologic, and bacterial studies were made. The histologic studies revealed slight to no inflammation in teeth treated with tricresol, sulfathiazole, beechwood creosote, and penicillin and a more severe inflammation in teeth treated with propyl mercuric chloride, pyridyl mercuric chloride, and sulfathiazole. The controls presented a histologic picture of osteoclasis with no inflammation to acute inflammation with repair. Robinson and Koch also reported that although their histologic studies offered some means of evaluating the tolerance of tissue to the drugs they tested, these studies were limited in value because of the lack of uniformity both in the microscopic appearance of the controls and in the tissue reactions to similar drugs.

The only histologic examination of human periapical tissue with regard to compatibility was conducted by Ostby[24] in 1957. Only one drug—EDTA (ethylenediaminetetraacetic acid)—was selected for study. This drug was tested on both vital and nonvital teeth. The pulp tissue or remnants of pulpal tissue were completely removed in both cases, and the canal and periapical tissues were

exposed to liberal applications of EDTA. The root canals were then hermetically obturated, so that the vital teeth were underfilled and nonvital teeth slightly overfilled. After observation periods varying from one to fourteen months, the apex of each tooth, together with its supporting structures, was surgically removed and examined histologically. Teeth which had undergone vital pulp extirpation showed clot formation with organization in the region of the apical foramen in the initial stages and deposition of fibrous connective tissue and hard tissue in the later stages. The necrotic teeth, all of which had been observed for thirteen to fourteen months, displayed regenerated alveolar bone and active bone formation with reorientation of functional periodontal fibers attached to newly formed cementum. Ostby concluded that both phases of this study confirmed the tolerance of this drug by the periapical tissues.

In 1958, Rubbo and associates[30] studied the reaction of tissue to root canal drugs in rabbits. In accordance with a technique described by Gronberg[36] in 1935, they injected 0.1 ml. of the test solution subcutaneously into the rabbit's ear. The drug's effect on the tissues was evaluated by the degree of hyperemia, edema, necrosis, and ulceration evident after a period of seven days. They found that camphorated parachlorophenol, beechwood creosote, formocresol, and oxpara liquid produced severe necrosis with ulceration. No reaction was noted with ATF (neomycin, bacitracin, polymyxin, A.163, noradrenalin, Sorbitol, and sterile water). In testing the tissue toxicity of ATF on human subjects in which 1 ml. of the drug had been injected intradermally, they found no evidence of hyperemia or edema after a period of twenty-four hours. Rubbo and colleagues[30] also observed the effects of ATF and other preparations on the morphology of the cellular elements of whole blood. Camphorated parachlorophenol, formocresol, and beechwood creosote reportedly produced instantaneous denaturation and coagulation of blood and complete disintegration of the leukocytes. ACB (aminacrine, cetrimide, and hydrobenzoates), formulated by Sullivan and Jolly,[35] and ATF caused hemolysis of red blood cells but had no effect on the polymorphonuclear elements.

Rabbits were also used as experimental subjects in the research undertaken by Schilder and Amsterdam.[31] In the first phase of their investigation 0.1 c.c. of each drug was injected intradermally into the abdomen of a rabbit. This was followed by an intravenous injection of trypan blue, a vital stain. Through visual examination after periods of one and three days, they observed that severe inflammation was induced by beechwood creosote, chlorinated soda, Azochloramid, camphorated parachlorophenol, eugenol, and formocresol. Cresatin and Grossman's[14] PBSC (penicillin, bacitracin, streptomycin, and sodium caprylate in a silicone fluid base) induced only a slight inflammation. Distilled water, silicone fluid, Zephiran chloride (1:1,000), and 3 per cent hydrogen peroxide produced no inflammation whatsoever. In the second phase of the investigation by Schilder and Amsterdam, 0.15 c.c. of each drug was introduced into the conjunctival sac of the eye. Physiologic saline solution was used here as a control. On the basis of swelling and hyperemia of the conjunctiva, clouding of the cornea, loss of clarity of normal anatomic markings, and external swelling

and exudation, beechwood creosote, Azochloramid, camphorated parachlorophenol, eugenol, PBSC, and formocresol caused severe inflammation; chlorinated soda and 3 per cent hydrogen peroxide caused moderate inflammation; Zephiran chloride (1:1,000) caused slight inflammation; and no inflammation was associated with the use of distilled water, Cresatin, and silicone fluid.

Finally, numerous articles[1, 2, 4, 12, 16, 24] dealing with the compatibility of living tissue to root canal medicaments as determined by subjective analysis of the degree of postoperative discomfort experienced by the patient following clinical administration of one of these agents have also been presented. Although it is significant to mention that such a method of evaluation has been used, a reiteration of the drugs tested and the results obtained is not deemed essential and therefore has not been included in this review.

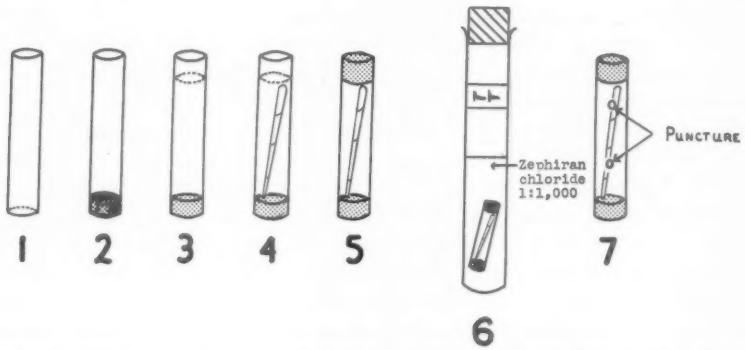

Fig. 1.—Diagrammatic representation of preparation of polyethylene Carpule prior to implantation in dorsum of hamster.

METHODS AND MATERIALS

The initial phase of the current study consisted of constructing a vehicle whereby a fixed amount of a test drug could be implanted subcutaneously in the hamster and allowed to diffuse through an opening of a given size into the surrounding tissues. For this purpose, 10 mm. lengths of Adams PE-160 (inside diameter 1.1 mm., outside diameter 1.5 mm.) Intramedic polyethylene tubing were selected. These were sealed to a depth of 0.75 mm. at one end with Caulk Grip, a quick-setting acrylic cement.

After the cement had set, a 27 gauge Luer-Lok syringe was used to fill the tube through its open end with the test drug. Care was taken to ensure that no air bubbles were entrapped in the tube as the test drug was added. A 7 mm. section of a sterile fine absorbent point previously moistened with the solution to be tested was also placed in the tube. Then, with a large sterile absorbent point, 0.75 mm. of tubing at the open end was thoroughly dried and sealed with acrylic cement. Prior to the surgical phase of the investigation, the outer surface of the polyethylene Carpule was disinfected by immersion for twenty-four hours in 1:1,000 Zephiran chloride. Sterilization in a dry-heat oven or

autoclave, though desirable, was not possible because of the deleterious effect that it would have on the polyethylene tubing as well as on the chemical structure of the drug to be tested. The steps involved in this phase of the study are shown in Fig. 1.

Young male and female Syrian hamsters were selected for the surgical implantation phase of the investigation. The surgical procedures that we used were suggested by Costich.[6]

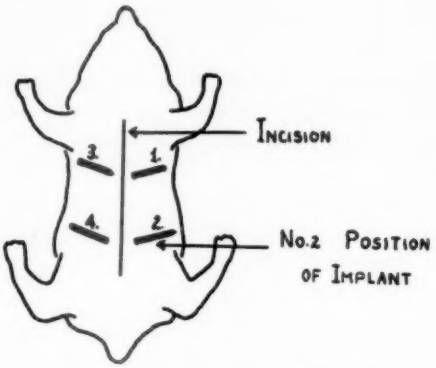

Fig. 2.—Surgical implantation of Carpule containing test solution into dorsum of hamster.

The animals were anesthetized by intraperitoneal injections of 1 c.c. of pentobarbital sodium (Nembutal) diluted in 4.5 c.c. of distilled water; 0.05 c.c. was administered for every 10 grams of body weight. The dorsum of each animal was then shaved, washed, and dried. An incision was made through the epidermis along the midline of the dorsum, extending 5 mm. above the lower border of the thoracic cage superiorly and opposite the upper border of the outstretched hind leg inferiorly. Next the epidermis was gently separated from the underlying tissues, and one Carpule was implanted in each of the four quadrants of the hamster's back (Fig. 2). Prior to insertion, each Carpule was thoroughly dried with sterile gauze and then punctured twice by slow rotation of a sterile 22 gauge syringe needle through the polyethylene. After the Carpule was positioned, the incision was sutured and the animal and the area into which each drug had been placed were recorded.

In this second phase, general principles of surgical technique were considered. All instruments used were sterilized by autoclaving prior to use, and the incision, implantation, and suturing were done quickly and gently to avoid unnecessary surgical trauma which might produce undesirable pathologic changes in the tissues.

A total of ten drugs were tested. These included camphorated parachlorophenol N.F., beechwood creosote N.F., eugenol U.S.P., liquefied phenol U.S.P., formaldehyde-cresol, Cresatin (Merck Sharp & Dohme), 1,2,3 solution (1 part beechwood creosote, 2 parts phenol, and 3 parts eugenol), Grossman's PBSC

(a polyantibiotic preparation combining potassium penicillin G, bacitracin, streptomycin calcium chloride complex, sodium caprylate, and silicone liquid DC 200), Ostby's EDTAC (a chelating and sterilizing agent for root canals, containing ethylenediaminetetraacetic acid and Cetavlon), and an aqueous solution of potassium penicillin G (50,000 units dissolved in 1 drop of sterile water). Physiologic saline solution was used as a control.

Following testing periods of both forty-eight and ninety-six hours for each drug, the animals were killed and the Carpules, along with the surrounding tissues, were dissected out in block form and prepared for pathologic examination.

Each drug was tested twice for both the forty-eight- and the ninety-six-hour periods. Thus, in all, forty-four tissue specimens were obtained. A total of twelve animals were used—six for the forty-eight-hour tests and six for the ninety-six-hour tests.

The tissue sections were prepared according to the method employed by the Pathology Department of the University of Michigan Dental School. Briefly, this consisted of the following steps:

1. Fixation in 10 per cent formalin.
2. Dehydration and clearing with alcohol and xylol.
3. Embedding in paraffin.
4. Sectioning (3 to 6 microns in thickness) at right angles to carpule.
5. Staining by the molasses plate and celloidin sheet method* with Harris' hematoxylin and eosin stain (bluish).
6. Mounting.

*The molasses plate method was described by A. S. Warthin in the May, 1924 issue of the *Journal of Laboratory and Clinical Medicine*. This method permitted serial sections to be transferred to a celloidin sheet, whereby they could all be stained simultaneously.

Fig. 3.—*A*, Photomicrograph illustrating the relationship of the control Carpule containing physiologic saline solution to the surrounding tissues. The duration of the implant was ninety-six hours. The large, clear, circular area in the center of the photograph represents the area occupied by the polyethylene tube. Preparation of the section for histologic examination resulted in a dissolution of the polyethylene. About the periphery of the space occupied by the tube, there is an encapsulation by fibrous connective tissue. This is composed of both young ovoid fibroblasts and more mature spindle-shaped fibroblasts. Several thin-walled blood vessels and the occasional inflammatory cell are also seen here. The area on the upper border of the space, where the periphery of the tube appears to lose continuity, is the site of puncture. The muscle and connective tissue surrounding the implanted Carpule appear normal and show no evidence of inflammation. (Hematoxylin and eosin stain. Magnification, ×35; reduced ⅝.)

B, Higher-power photomicrograph of the puncture site of Carpule containing physiologic saline solution which was implanted for ninety-six hours and outlined in *A*. Numerous young fibroblasts are seen in this area. The occasional inflammatory cell is present, but this is restricted to the area immediately surrounding the puncture site. The muscle tissue approximating the puncture appears normal and shows no evidence of inflammation. (Hematoxylin and eosin stain. Magnification, ×90; reduced ⅝.)

C, Higher-power photomicrograph of the connective-tissue capsule about the space occupied by polyethylene Carpule which contained physiologic saline solution and which was implanted for a period of ninety-six hours. This photomicrograph was taken, as shown in *A*, along the tube border a short distance from the puncture site and shows quite clearly the young and mature fibroblasts of which the connective-tissue capsule is composed. In the upper portion of the capsule, one large thin-walled blood vessel and several small blood vessels are seen. The occasional inflammatory infiltration is evident, but this is restricted to the tissues immediately surrounding the space. The approximating muscle tissue and muscle fascia appear normal and show no evidence of inflammation. (Hematoxylin and eosin stain. Magnification, ×240; reduced ⅝.)

Fig. 3.—*(For legend, see opposite page.)*

To ensure a more accurate comparison of the differences in the microscopic effect of the test drugs on the tissues, serial sections of each specimen were made. Of these, the section demonstrating the most severe reaction about the area of the puncture was selected for more detailed examination and evaluation.

The relationship of the tube to the surrounding tissue in a stained pathologic section is shown in Fig. 3.

FINDINGS

After forty-eight hours, the tissue into which the control Carpule containing sterile physiologic saline solution had been implanted demonstrated a proliferation of young fibroblasts and endothelial tissue about the periphery of the polyethylene tube. Several blood vessels containing red blood cells were evident in this area, as well as a mild infiltration of neutrophilic polymorphonuclear leukocytes and monocytes. The latter was caused by the slight degree of mechanical trauma involved in the surgical phase of the investigation. No concentration of polymorphonuclear cells or any evidence of necrosis was seen in the area adjacent to the puncture.

At the end of forty-eight hours, the phenol-treated tissues demonstrated little proliferation of reparative elements about the border of the tube. There was a purulent exudate in the area near the tube opening as well as a diffuse, active, inflammatory infiltration involving muscle, adipose tissue, and connective tissue. Hyaline degeneration was evident in the fascial planes opposite the tube opening, with some simple necrosis of the voluntary muscle itself. A moderate-sized vein and several smaller blood vessels in the surrounding area had undergone thrombosis.

In the tissues subjected to formocresol there was a purulent exudate at the border of the tube with an intense diffuse, active inflammation involving adipose tissue and the fascial planes of the voluntary muscle after forty-eight hours. Necrosis of connective tissue had occurred near the puncture area of the tube.

The forty-eight-hour beechwood creosote section showed a narrow zone of necrosis around the tube, with a heavy infiltration of inflammatory cells and focal abscess formation near the tube opening. There was also a moderate but diffuse spread of leukocytes into the fascial planes and subcutaneous tissue.

The reaction to the 1,2,3 solution was similar to that produced by the beechwood creosote, but it was not so intense.

After forty-eight hours, camphorated parachlorophenol and Cresatin, though both diffuse in their action, were considerably less irritating than any of the previous four drugs. The camphorated parachlorophenol caused a moderate infiltration of leukocytes, monocytes, and lymphocytes about the border of the tube, with no evidence of tissue necrosis present at the area of puncture. A moderate, somewhat diffuse, active subacute inflammation was present in the surrounding connective tissue. A heavier inflammatory infiltration about the tube border was noted in the section injected with Cresatin. The involvement of the surrounding connective tissue was less intense, however, although it was

more extensive. A proliferation of fibroblasts and endothelial tissue about the periphery of the tube opposite the puncture was present in both sections.

In forty-eight hours eugenol and PBSC produced reactions which were localized to the puncture area of the tube. With the PBSC there was a purulent exudate with a slight zone of necrosis concentrated about the opening. A mild, active, subacute inflammation was present in the connective tissue approximating this area. With eugenol, suppuration and a more intense inflammatory infiltration of the surrounding tissue were noted. In both specimens an attempt at regeneration was seen along the opposite border of the tube. Although localized in their action, these drugs appeared to be more irritating than either Cresatin or camphorated parachlorophenol.

Penicillin and EDTAC were the least irritating in their action after forty-eight hours. Only a mild inflammatory response localized to the area of puncture was evident in both instances. A very small area of necrosis was also noted in this region in the specimen injected with penicillin. Normal healing had not been interrupted, however, as evidenced by the fibroblastic proliferation about the tube.

Following a test period of ninety-six hours, a connective-tissue capsule containing mature, young, and proliferating fibroblasts as well as several thin-walled capillaries was seen about the control Carpule which contained the sterile physiologic saline solution. Some inflammation was still present, but this was very mild and was confined to the tissue immediately surrounding the tube.

After ninety-six hours, a zone of coagulation necrosis about the tube opening was seen in the section subjected to phenol. In addition, hyaline degeneration of connective tissue and a moderate inflammatory infiltration involving both connective tissue and muscle were evident in the tissues some distance from the tube.

An extensive, active, subacute inflammation about the tube and extending out into the surrounding adipose tissue and muscle fascia was still present after ninety-six hours in the section subjected to formocresol. Some coagulation necrosis of connective tissue was also noted in the area adjacent to the puncture.

Beechwood creosote induced an abundant purulent exudate about the tube and a diffuse, intense, subacute inflammation in the surrounding connective tissue and muscle fascia for some distance beyond. Evidence of tissue necrosis was seen about the actual point of puncture.

A purulent exudate was also seen in the specimen subjected to the 1,2,3 solution. Again, this drug appeared to be less irritating than the beechwood creosote, since the amount of tissue necrosis was less extensive. Furthermore, the inflammation, though similar in distribution, was more moderate in intensity.

The reaction to eugenol observed after ninety-six hours was less localized than that seen at the end of the forty-eight-hour period. A moderate, subacute inflammation was now present in the adjacent connective tissue. A moderate zone of connective-tissue necrosis and an initiation of muscle-tissue degeneration were evident in close proximity to the puncture area. Some proliferation of fibrous connective tissue along the sides of the tube had occurred.

PBSC caused a focal accumulation of leukocytes about the surface of the tube, with some simple necrosis of tissue at the area of puncture. As in the specimen subjected to eugenol, inflammation to a moderate degree was seen extending a limited distance into the surrounding adipose and fascial tissues.

Sections in which camphorated parachlorophenol and Cresatin had been implanted showed no evidence of tissue necrosis and an active condensation of young connective tissue about the periphery of the Carpule away from the site of opening. Both sections elicited a concentration of polymorphonuclear cells at the puncture area and a moderate inflammatory response which extended into the tissue for some distance beyond the tube. The focal accumulation of leukocytes appeared slightly greater in the specimen subjected to parachlorophenol.

The penicillin solution and EDTAC again proved to be the least irritating of the drugs tested. The inflammation present in specimens subjected to these drugs was mild in nature and was restricted to the tissue immediately approximating the tube. Fibroblastic encapsulation was seen in sections subjected to both drugs, and in the EDTAC specimen some granulation tissue composed of proliferating young fibroblasts and endothelial tissue and some lymphocytes were still present at the border of the tube.

DISCUSSION

Chemical injury occurs when living tissue is exposed to drugs which are not compatible with cellular metabolism. The injury may be mild, in which case there will be only an alteration in the metabolism and function of the cell to compensate for the presence of the irritant. On the other hand, as pointed out by Forbus,[10] the irritation may be more violent, producing a complete cessation of all cellular activity, with death of the cell and total loss of its contribution to the cell community. With the milder form of irritation, in which the normal metabolism of the cell is interrupted, microscopic evidence of injury to the cell is not always evident. However, when the physiologic tolerance is exceeded, an inflammatory response in the body is elicited and, as pointed out by Menkin,[21] is indicative of the cellular injury. This response is defensive in nature and serves the purpose of localizing and disposing of the injurious agent to protect the tissues against further damage. Moreover, Menkin has shown that this phenomenon is manifested by all higher animals and that it tends to follow a uniform pattern, regardless of whether the irritant is of a chemical, electrical, bacterial, or mechanical type.

The tissue reaction to root canal medicaments as encountered in the clinical practice of endodontics is influenced by seven factors: (1) the type, concentration, and physical form of the drug employed, (2) the amount or volume of the drug used, (3) the manner in which the drug is placed and sealed into the root canal, (4) the size of the apical foramen, (5) the histologic status of the periodontium, (6) the length of time for which the drug is exposed to the tissues, and (7) the individual patient's susceptibility to injury.

The type and concentration of the drug employed is perhaps the most obvious of these factors. Chemical agents which have a high irritating potential and which are used in high concentrations on living tissue produce severe cellular injury. Conversely, when a lesser concentration or a less irritating drug is used, the injury produced is not so intense. This has been exemplified by the differences in the degree of tissue irritation produced by the various agents tested in this investigation. References to this effect have also been cited by the other investigations[3, 13, 25, 29, 30, 31] who have conducted research in this field. Logically, it follows that the volume of the drug and the length of time that it is exposed to the tissues similarly influence the proportionality of the irritation. The ability of drugs to coagulate protein also appears to alter the extensiveness of their action. This was shown by Coolidge's[5] study in which the noncoagulating drugs penetrated deeper into the tissues than the drugs that coagulated protein.

The physical form of the drug and the size of the apical foramen effect the rate at which diffusion into the periapical tissues occurs. Drugs which diffuse slowly have a lower tissue concentration and are less toxic in their action, since they are more readily diluted and destroyed by the body's defenses. Consequently, Forbus[9] has stated that chemical irritants which are in a semisolid or paste form and which must first be dissolved in the tissue fluids before diffusion can occur are less injurious than those which are already in solution. Furthermore, Frobisher[11] has shown that among the liquids the lower the surface tension, the greater the diffusive ability. Therefore, when irritating medicaments are sealed into the root canal, a more extensive involvement of the periapical region is encountered when liquid drugs of low surface tension are used. The involvement produced by higher-surface-tension liquids or drugs in semisolid or paste form is less extensive due to the reduced rate of diffusion. In both cases, increasing the size of the apical opening will accelerate the rate at which diffusion proceeds.

The extent to which the drug infiltrates into the periodontium will also be influenced by the histologic status of the tissues. Menkin[23] has shown that localization of an irritant occurs more readily in an inflamed area than in normal tissue. This, he feels, is primarily referable to the occlusion of lymphatics and blood vessels present in the pre-existing inflammation.[21] Therefore, clinically, the involvement of the periapical tissues will be less extensive when an inflammatory reaction is already present. It should be remembered, however, that the superimposition of injury on already damaged tissue may also lead to focal necrosis and a severe pericementitis.

The need for care in the placement and sealing of germicidal agents into the root canal has already been stressed by Sommer and associates.[32] Improper placement of the medicaments in the canal may cause the agent to be forced mechanically through the apical foramen, and the degree of tissue damage may be magnified considerably. This is encountered more frequently when a syringe or an endodontic plugger is employed for this purpose. An exaggerated reaction may also occur when irritating drugs are sealed in the canal under pressure, resulting in an accelerated rate of diffusion.

Local tissue response to chemical irritation is further influenced by individual differences. The reactions produced will not only vary with differences in the resistance of individual patients to injury but also, as pointed out by Forbus,[9] with their ability to destroy, fix, eliminate, or otherwise remove the harmful substance from the sphere of action.

Likewise, the various tissues of the body will show different reactions to similar irritants. Surface epithelium, for example, has a greater resistance to injury than does young connective tissue. The epithelial cornea of the eye, on the other hand, is more irritable than either surface epithelium or young connective tissue. Even among similar tissues, variations in sensitivity exist. This is essentially due to differences in the degree of metabolic activity. In general, the lesser the metabolic rate (such as that seen in old, more mature tissue in comparison with younger proliferating tissue), the greater the resistance to injury.

The investigative technique employed in this study was designed to control as closely as possible the conditions which are encountered clinically in the use of root canal medicaments.

The type, concentration, and physical form of the test preparations were those commonly used in the treatment of septic root canals.

The volume of the test drug was controlled by the use of standard-sized polyethylene Carpules. Discounting the seal, this was mathematically calculated to be 0.008 c.c. An earlier investigation by Stewart[33] had shown this to be the average capacity of the mandibular central and lateral incisors after enlargement with a No. 5 endodontic file. A volume of 0.016 c.c., which Stewart had estimated as the average capacity of the maxillary central incisor, was originally desired, but it was found that, after implantation, mechanical irritation to the tissues was produced when movements of the test animal caused a shifting in the position of a larger Carpule. The size of the Carpule was therefore reduced at the expense of the volume in order that it might be compatible with muscular activity.

Diffusion of the drug through the apical foramen or foramina was simulated by careful puncturing of the tube prior to its implantation. When a 22 gauge syringe needle was used for this purpose, an opening averaging 540 microns was produced. Measurements made by Kuttler[18] on 402 extracted teeth showed that this diameter approximated that of the average apical foramen encountered among the 18- to 25-year age group. Theoretically, therefore, the punctured polyethylene Carpule could be considered analogous to the mandibular central or lateral incisor of an 18- to 25-year-old person.

Syrian hamsters were selected as test animals since, through extensive inbreeding, the individual differences in their reactions to similar irritants were so slight[7] that they could be considered insignificant. Slight variations in the reactions were encountered, however, as a result of implantation of the Carpule in different areas of tissue. In sections demonstrating an abundance of adipose and muscle tissue, the inflammatory reaction was less intense than in sections containing highly vascular young connective tissue. Significantly,

though, the pattern of reactions cited in the findings was not greatly altered. That is, where necrosis of tissue was produced in one instance, it was produced in all instances, despite the area of implantation. Only slight differences in the degree of severity were noted. Similarly, where extensive inflammation but no necrosis was produced, only slight differences in the intensity of the inflammation resulted. Although the variations arising from this situation could not be overlooked, it was felt that they were not of sufficient magnitude to hinder the evaluation of the test drugs with respect to their suitability for use as bactericidal agents in endodontic therapy.

When an investigative technique such as the one described here is undertaken, consideration must be given to the "hollow tube effect" studied by Rickert and Dixon[26] and discussed in a later publication by Sommer, Ostrander, and Crowley.[32] This effect is seen in the irritation produced by stagnation and decomposition of tissue fluids within an implanted hollow tube and the eventual release of these degradation products into the surrounding tissue. After a period of ninety-six hours our control specimens showed no significant evidence of tissue irritation, either from this effect, from the polyethylene, or from the mechanical trauma of the surgical procedures. It is assumed, however, that if the tube had been allowed to remain for a longer time some irritation from tissue fluid decomposition would have been produced. A test period of forty-eight hours was adopted in our investigation of whether any relatively immediate effects on the tissue were produced and the extent, if any, to which the tissues were involved. Second, the adoption of two test periods afforded an excellent opportunity of determining how the pattern of drug action might be influenced by a time factor. Examples of this time effect were observed in the actions of phenol, PBSC, and eugenol. After a forty-eight-hour period the phenol was shown to produce widespread tissue injury, as evidenced by the degree of degeneration, necrosis, and inflammation present microscopically. Observations made after ninety-six hours revealed that the zone of cellular injury had not become appreciably greater and that coagulation necrosis had occurred about the tube opening where the concentration of the drug in the tissues was the greatest. In a sense, the precipitation of protein which accompanies this necrosis is a defensive phenomenon, as cited earlier by Coolidge,[5] in that it impedes further diffusion of the drug into the tissues. With regard to the latter two drugs (PBSC and eugenol), their action after forty-eight hours, although moderately destructive, remained relatively localized to the area of puncture. Allowing the implanted drug to remain in the animal for an additional forty-eight hours permitted a more extensive diffusion of both agents into the surrounding tissues, which caused a greater zone of tissue irritation.

In analyzing the results obtained from this investigation, it is apparent that all of the agents tested, with the exception of the sterile physiologic saline solution, proved irritating to hamster tissue. Differences occurred only in the degree of irritation recorded. Since the agents tested represented a large percentage of the medicaments commonly employed in the sterilization of root

canals, the rationale for their use with respect to tissue tolerance appears to be not whether the drug is nonirritating but, rather, the degree to which it is irritating. EDTAC and the penicillin solution, for example, produced only a mild reaction and did not interfere with normal healing. It is also interesting to note that one very small area of simple necrosis was observed at the puncture area near the Carpule containing penicillin where, as mentioned earlier, the concentration of the drug in the tissues is greatest. Since the solution was found to have a pH of 5.5 and was highly hypertonic, this effect was believed to be caused by the inability of the body fluids to buffer it adequately at this point.

Phenol, formocresol, beechwood creosote, and 1,2,3 solution, on the other hand, were found to produce extensive and severe cellular damage with tissue necrosis. As would be expected, clinical use of some of these drugs has led to a higher incidence of pericementitis[3, 25, 31] than is encountered when milder, less irritating preparations, such as Cresatin, camphorated parachlorophenol, or even PBSC, are used. Other effects, such as a prolonged rate of healing following the application of these agents and the possibility of establishing an area of low resistance or locus minoris resistentiae, as referred to by Menkin,[22] which could lead to facilitated growth of infective bacterial elements, as cited by Robinson and Koch[29] and Macdonald and Hare,[19] also serve to demonstrate why the use of the less toxic medicaments remains more desirable.

Finally, in order to keep sterilization by chemotherapeutic methods in its proper perspective, we should remember that compatibility with living tissue is only one of the desirable characteristics of root canal medicaments. As summarized by Ostrander and his colleagues[32] and by Stewart,[33] it is important that these drugs also meet the following requirements:

1. They must be rapidly effective in eliminating or destroying those microorganisms normally found in the root canal.
2. They must be effective in destroying, neutralizing, or eliminating any toxic products which may be present in these canals.
3. They must be nonstaining to tooth tissue.
4. They must have good penetrating quality in order to be effective deep within the dentinal tubules.
5. They must remain stable at room temperature for long periods of time.
6. They must not be inactivated by blood, serum, protein, pus, or other organic matter which may be present within the root canal or periapical tissues.
7. They must be readily available.
8. They must be easy to use.

An additional requirement stressed by Sommer, Ostrander, and Crowley[32] and also by Grossman[15] is that the drug should in no way interfere with an accurate culture technique. Unfortunately, none of the root canal antiseptics now available to the dental profession possess all of these characteristics. Until

an ideal drug is introduced, the choice of a root canal antiseptic should be restricted, as postulated by Ostrander,[32] to those agents which come closest to meeting the requirements of the ideal.

SUMMARY

The reaction of hamster tissue to various drugs now being used in the sterilization of root canals was investigated. The method employed consisted of surgical implantation of punctured polyethylene Carpules containing the medicaments into the dorsum of the test animal. It was felt that with this technique conditions encountered in the clinical use of the drugs could be simulated. The drugs tested included phenol, formocresol, beechwood creosote, 1,2,3 solution, PBSC (a polyantibiotic paste), eugenol, camphorated parachlorophenol, Cresatin, a saturated solution of 50,000 units of water-soluble potassium penicillin G, and EDTAC (a newer agent, recently introduced by Ostby, which contains ethylenediaminetetraacetic acid and cetavlon). Sterile physiologic saline solution was used as a control. Following test periods of forty-eight and ninety-six hours the tissues were excised, prepared, and examined histologically.

As explained in the discussion, the reactions encountered clinically with the use of root canal medicaments are influenced by (1) the type, concentration, and physical form of the drug, (2) the volume of the drug, (3) the manner in which the drug is sealed into the pulp canal, (4) the size of the apical foramen, (5) the histologic status of the periodontium, (6) the length of time the drug is exposed to the tissue, and (7) the susceptibility of the individual to injury. Drugs that are highly irritating and cause extensive damage produce a higher incidence of pericementitis and a reduced rate of healing. Furthermore, there is evidence that they may also lead to the establishment of anachoretic infection.

CONCLUSIONS

This investigation has shown that although most of the root canal antiseptics currently employed in endodontics are irritating to living tissue, some of these medicaments can produce relatively extensive and severe cellular injury. Since in endodontic therapy, as in any other form of dental therapy, it is desirable to achieve successful results while subjecting the patient to the least possible amount of trauma, the use of the more irritating drugs as sterilizing agents should be avoided. Although no bacterial tests were undertaken in this study, experimental evidence[15, 32] indicates that many of the preparations which are more readily tolerated by the tissues are also more effective in a germicidal capacity. This tends only to re-emphasize why preference is given to the less-irritating medicaments.

Finally, as mentioned earlier in this article, a truly nonirritating agent that meets all the requirements deemed essential in a root canal antiseptic has not yet been introduced. The need for such an agent is evident, and until one is discovered research in this field should be continued.

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ABERRANT SALIVARY GLAND DEFECT IN THE MANDIBLE

Report of a Case

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ECTOPIC salivary gland tissue is one of the causes of well-circumscribed, benign-appearing, ovoid, radiolucent lesions found in the body of the mandible near the angle. Thoma[1] described such a defect which was caused by a blood-vessel aneurysm. Many other lesions have not been surgically explored and were labeled static bone cavities.[2-6, 9] A number which have been explored were found to contain glandular tissue.

Stafne[2] described thirty-four patients (one with bilateral lesions) in none of whom was exploration attempted. Five of the lesions were followed roentgenographically for as long as eleven years without detectable change. Stafne observed that the similarity in x-ray appearance and the consistent location of these bone cavities suggest a definite entity, probably with a common etiological factor. Jacobs[5] mentioned a case of submaxillary gland within a "mandibular embryonic defect." Fordyce,[6] reporting two cases in which a submaxillary gland was found within a mandibular recess, suggested the use of submandibular sialography.

Of the illustrated cases in the literature, the only lesion which appears to have been situated elsewhere than below the mandibular canal in the third molar region was one reported by Richard and Ziskind.[7] That lesion was located in the body of the left mandible, above the mandibular canal and in the region of the apices of the cuspid and premolars. Tissue examination revealed a mixed glandular structure resembling sublingual gland.

Choukas and Toto[8] described a case in which a lesion proved to be a developmental submaxillary defect of the mandible. They attributed the defect

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in this case to hypertrophy of the submaxillary gland or to a congenital defect resulting from entrapment of gland tissue. They also suggested that in those cavities which were surgically explored and found to be devoid of contents entrapped glandular tissue may have been dislodged during the operation, which seems entirely possible.

CASE REPORT

A 50-year-old Filipino man was found, on routine dental roentgenographic examination, to have a radiolucent lesion below the molar teeth in the right mandible. Three extraoral projections of the mandible demonstrated what appeared to be an osteolytic defect of the body of the mandible along its inferior margin and below the mandibular canal. The defect measured 2.2 by 1.5 cm. Its margins were clearly defined and were comparable in density to cortical bone. Biochemical examinations, including calcium, phosphorus, and alkaline phosphatase determinations, were within normal limits. Roentgenographic examination of the skull showed the cranial bones to be of normal contour, outline, thickness, and density.

Fig. 1.—Preoperative view of osteolytic defect of body of right mandible below mandibular canal.

On June 23, 1959, after inferior alveolar and buccal block anesthesia had been obtained with lidocaine hydrochloride, a biopsy of the lesion was attempted through the buccal vestibule subjacent to the molar region. After a small quantity of tissue had been removed, considerable hemorrhage ensued and prevented further exploration. Frozen section and permanent hematoxylin and eosin sections revealed only loose fibroadipose tissue. The intraoral biopsy site was allowed to heal.

On July 16, 1959, following induction of general anesthesia, the defect was approached by the submandibular route. Exposure of the mandible as far as the original biopsy site revealed that the lingual cortex was depressed and that the tissue within the defect was continuous with the superior pole of the submaxillary gland. The tissue occupying the bony defect was excised, and autogenous cancellous bone chips were packed into the bone defect. Recovery was uneventful.

Fig. 2.—Preoperative lateral view. Note that the inferior margin of the mandible is intact.

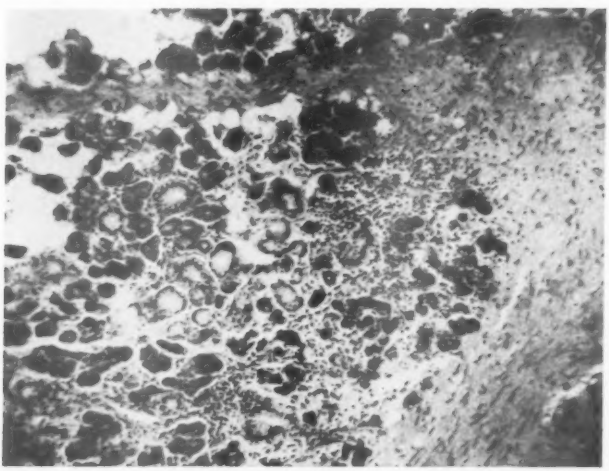

Fig. 3.—Margin of lesion demonstrating fibrous tissue infiltrated by round cells at right; salivary gland at left.

Pathologic Findings.—Gross examination revealed a 2 by 1.2 by 1.2 cm. yellow-tan mass, made up of a mixed compound tubuloalveolar gland. Its alveoli were predominantly serous. Heavy fibrous tissue infiltrated with round cells was found at the gland margin, compatible with a reaction to the previous surgical procedure. This submaxillary gland tissue contained no neoplasm.

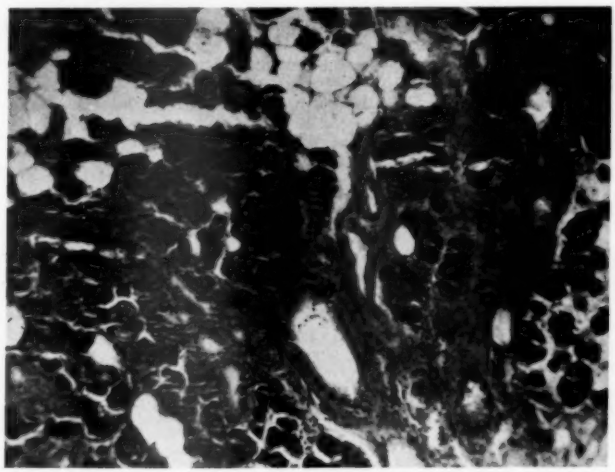

Fig. 4.—Structure of submaxillary gland.

DISCUSSION

It seems universally agreed that surgical intervention is not indicated if these cavities can be shown to represent normal gland tissue. It would seem that in suspected lesions of this nature diagnostic measures should include (1) submaxillary sialography; (2) an axial skull roentgenogram or an occlusal view of the mandible to show the lingual surface of the mandible; and (3) where exploration of the defect is desired, a lingual mucoperiosteal flap and detachment of a portion of the mylophyoid muscle to demonstrate the impingement of gland tissue upon bone.

CONCLUSIONS

1. Some cystlike defects of the body of the mandible have been shown to be occupied by normal salivary gland tissue.

2. Surgical intervention does not appear to be indicated.

3. Sialograms and special projections to demonstrate the lingual cortex of the mandible should be employed. It is suggested that reflection of a lingual mucoperiosteal flap and detachment of a portion of the mylohyoid muscle should demonstrate these defects if all other measures fail to do so.

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REDUCTION OF A ZYGOMATICOMAXILLARY COMPLEX FRACTURE BY THE ANTRAL BALLOON TECHNIQUE

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THE heaviest abutment of the facial skeleton is formed by the zygomatic bone.[1] Because of its anatomic position this bone is liable to traumatic injury and is the most commonly fractured facial bone, after the mandible and the nasal bones.[2] Traumatic injuries of the face rarely involve the zygoma alone but tend also to involve its articulations with other facial and cranial bones. Injuries of this type are classed as zygomaticofacial compound dislocations.[3]

DIAGNOSIS

The considerable ecchymosis and edema which follow facial injury of any kind make the diagnosis of a zygomaticofacial compound dislocation difficult. This is especially true when the zygomaticomaxillary complex and the orbital regions are involved. The initial inspection and palpation of this area may be misleading, and an incorrect diagnosis may follow. Accurate roentgenographic surveys are necessary if one is to avoid overlooking any fracture of this area. These should include postero-anterior projections, Waters' views of the sinuses and maxillae, and bregma-menton films of the zygomatic arches. Special techniques, such as laminography, may be used at the discretion of the operating surgeon.

The classic signs of fracture of the zygomaticofacial complex include edema and ecchymoses of the adjacent face and eyelids, subconjunctival hemorrhage, unilateral nasal hemorrhage, point tenderness over the areas of suture separation, and paresthesia or anesthesia of the involved infraorbital nerve. Impingement on the coronoid processes, with limitation of oral opening, may also be noted. Intraoral digital examination should be performed high in the buccal sulcus to uncover other abnormalities.

An important point to consider is that when there is an injury to the middle third of the face a concomitant basilar skull fracture may be present.[4] This should be suspected until it is ruled out by competent clinical neurologic examination. Surgical interference should never be undertaken prior to this clearance. Ophthalmologic examination is indicated to rule out enophthalmos and exophthalmos following disruption of the orbital floor and tissues. Fre-

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quently, this examination may lead to the discovery of a temporal lobe shift or increased intracranial pressure which must be given priority of treatment. Diplopia and disturbed muscular functions will usually be corrected by reduction of the displaced parts.

METHODS OF TREATMENT

A study of the literature reveals a multiplicity of methods for treating fractures of the zygomaticofacial complex.[5-11] Regardless of the treatment method to be employed, all complicating factors must be ruled out prior to

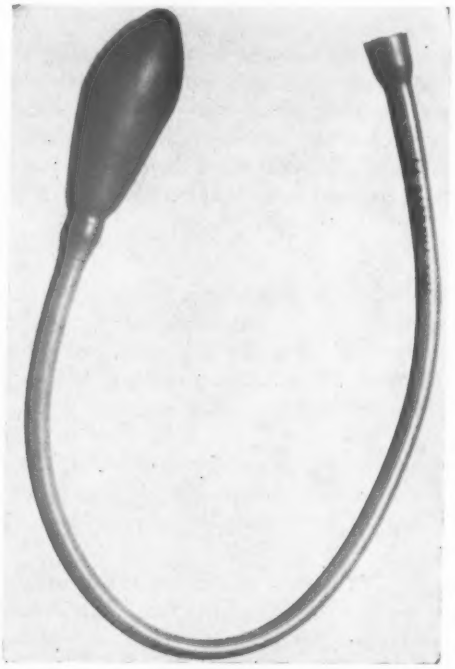

Fig. 1.—The Shea-Anthony antral balloon.

surgical interference. If no complications are present the ideal time for reduction and fixation of the fracture segments lies between twenty-four and seventy-two hours following the initial injury. Subsequent to this, organization of the blood clots and beginning fibrosis may preclude more conservative therapy.

The use of cumbersome appliances, either intraoral or extraoral is no longer essential. With the advent of antibiotics, it became possible to employ more conservative methods which are well tolerated by the patient and which promote excellent union of the distracted parts. For the competent oral surgeon,

the Shea-Anthony antral balloon (Fig. 1) is an ideal fixation appliance.[12, 13] Since it permits direct visualization of the parts to be reduced and fixed, and also since it is quite easy to handle, this antral balloon affords excellent results in the therapy of zygomaticofacial compound dislocations.

TECHNIQUE OF TREATMENT

In the majority of fractures of the zymaticofacial compound, there is concomitant antral involvement. This can range from simple hematoma formation to extensive crush injuries of the antral wall. Because of these findings, the antrum is explored; the removal of debris and blood clots and the more direct vision of the distracted parts made possible by such antral exploration promote prompt healing. Either local or general anesthetics may be used for this procedure.

In the technique to be presented here, the oral surgeon takes advantage of the antral exploration to introduce a latex balloon into the antral space. When inflated with sterile saline solution, this latex balloon produces pressure against the approximated bony fragments and maintains the parts in position during the healing stages.

An incision is made high in the mucobuccal fold over the canine fossa. The tissue is retracted and the antral wall is exposed. This wall can be intact, but in many cases is has been comminuted and may be perforated. An opening is made in the usual fashion and enlarged to permit easy instrumentation and vision of the antrum. The debris and blood clots are removed. By means of finger manipulation, the distracted parts are put back into position. It is important that the facial contours be thoroughly checked at this time to ensure as nearly a normal result as possible.

When the antrum is clear and the parts are in good approximation, an intranasal antrostomy is performed, either by enlargement of the antral meatus or by an incision under the inferior turbinate. The Shea-Anthony balloon is then introduced into the antral space, and its neck is brought out through the nasal aperture by way of the opening created by the antrostomy. Sterile saline solution, which I prefer to air, is injected into the balloon in an amount sufficient to elevate the approximated parts slightly beyond their normal position. The incised areas are then closed, and a nasal gauze plug is placed around the neck of the balloon.

The balloon is left in situ for one week. Then it is deflated by the withdrawal of 2 c.c. of saline solution every other day until all of the solution is removed. The deflated balloon is easily extracted through the surgically created opening in the antrum, and healing is uneventful.

If there is pressure pain during the initial healing period, it is easy to remove a sufficient quantity of saline solution from the balloon to alleviate symptoms. Under no circumstances should all the saline solution be evacuated at one time or the balloon removed prematurely, as the approximated parts can collapse prior to fibrosis and beginning bone formation.

CASE REPORT

On July 4, 1955, a 60-year-old white farmer was admitted to the Veterans Administration Hospital in Birmingham, Alabama. He complained of pain accompanied by swelling and discoloration in the left cheek and pain in the lower left leg. The patient stated that on the previous day he had fallen from a porch, striking his left side on an iron pipe. Since the accident the left side of his face had been severely swollen, and pain in his left leg had forced the patient to walk with a limp. He did not lose consciousness after the fall, and he had been able to see out of his left eye until it was closed by the swelling.

Fig. 2.—Waters'-view roentgenogram of the sinuses revealing a displaced, depressed fracture of the left inferior orbital rim.

Examination Findings.—Physical examination revealed a well-developed, 60-year-old white man with a swelling of the left face and limited motion in the left leg. Externally, there were periorbital ecchymoses of the left orbit and multiple abrasions of the upper left face. Edema in this area prevented adequate palpation of the infraorbital ridge. The left infraorbital nerve apparently had been injured by the blow, as was evidenced by paresthesia of the normal distribution. The left eyelids were edematous. The patient was edentulous and presented no intraoral evidence of trauma. The lower left leg was also ecchymotic and bruised. There was decided limitation of motion in the lower portion of this leg.

X-ray examination disclosed a recent depressed fracture of the left orbital rim. There was considerable diastasis of the fragments. Some clouding was seen in the left maxillary sinus (Fig. 2). A depressed fracture of the left zygoma was also observed, although depression was minimal. The bones of the cranial vault and base of the skull showed no evidence of fracture. Roentgenograms of the left ankle revealed a recent fracture of the lateral malleolus, with no significant displacement.

The impression gained at this time was that the patient had sustained fractures of the left zygoma, the inferior orbital rim, and the lateral malleolus of the left leg secondary to a fall.

All laboratory findings were within normal limits. A slight elevation in temperature, to 99.2° F., was noted on admission.

Treatment and Course.—At the time of the original admission on July 4, 1955, the dental service was put in charge of this case. The patient was placed on a regimen of procaine penicillin, 300,000 units twice daily, and sedatives were given as needed. After he had been skin-tested for possible sensitivity, he was given 3,000 units of tetanus antitoxin as well as 0.5 c.c. of tetanus toxoid.

Fig. 3.—The Shea-Anthony antral balloon in position in the left maxillary sinus.

Consultations with the neurologic, ophthalmologic, and orthopedic services were requested. The orthopedic service applied a boot cast to the left leg on July 5. The ophthalmologic consultation revealed no signs of injury, but the ophthalmologist requested that surgical intervention be delayed until July 7. The neurologic service noted no signs of cranial or spinal trauma and gave clearance for an operation.

On July 7, 1955, the patient was given Demerol hydrochlorides 75 mg., and atropine sulfate, 1/150 gr., intramuscularly one hour before the operation. In the operating room anesthesia was induced with Pentothal sodium and Anectine chloride. A nasoendotracheal tube was placed, and nitrous oxide–oxygen anesthesia was started. The patient was then prepared and draped in the usual manner, and the oropharynx was suctioned dry. Three 4 by 8 inch pieces of gauze soaked in sterile saline solution were packed around the endotracheal tube to prevent possible aspiration of blood, mucus, or other debris during the operation. Next, 2 per cent lidocaine hydrochloride (Xylocaine) containing 1: 100,000 epinephrine was infiltrated around the intraoral operative site. A large mucoperiosteal flap was elevated from the left lateral area to the upper left molar area,

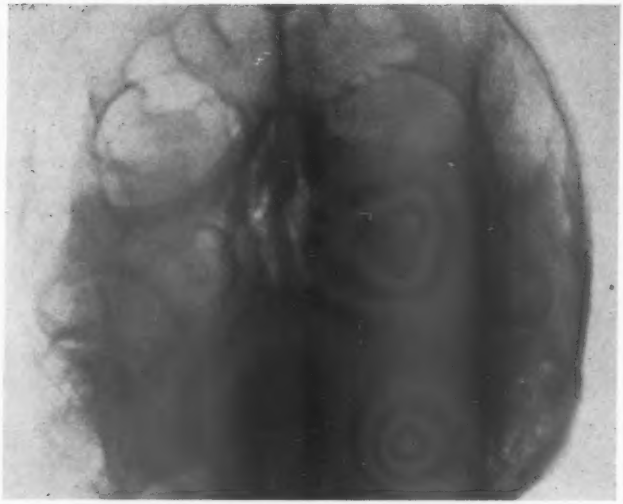

Fig. 4.—Postoperative Waters'-view roentgenogram of the sinuses showing relative opacity of the left antrum and repositioning of the fracture segments.

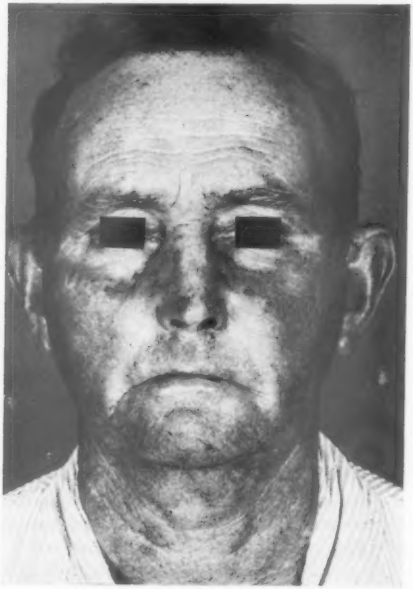

Fig. 5.—Postoperative photograph of patient following removal of the antral balloon. The contour of the left orbital rim is nearly normal, and there is no proptosis of the left eye.

exposing a large perforation in the antral wall. This opening was further enlarged with rongeurs forceps. Bleeding was profuse at this point, but it diminished greatly with the removal of the involved antral membranes. An intranasal antrotomy was performed below the inferior turbinate. The fracture segments were then repositioned manually until good approximation was obtained. A Shea-Anthony antral balloon was inserted through the intraoral incision, and the balloon catheter was brought out through the opening created by the intranasal antrotomy (Fig. 3). Approximately 12 c.c. of sterile saline solution was inserted into the balloon under pressure by means of a Luer-Lok syringe until light pressure was maintained against the fracture segments. This sufficed to retain the nearly normal contour of the left cheek and orbital areas. The mucoperiosteum was then reapproximated with 000 black silk sutures. A nasal gauze plug was inserted into the left nostril around the balloon catheter. The patient withstood the procedure well and was sent to the recovery room.

Fig. 6.—Roentgenogram taken six months after operation shows clearing of the left maxillary antrum and good healing of the left inferior orbital rim.

Postoperative Course.—Postoperatively, the patient did remarkably well. The penicillin regimen was continued for six days, but little medication for pain was used during the recovery period. An icecap was applied for the first ten hours. There was some edema in the operative area, but this was never a serious problem. Immediate improvement in facial contour was noted following the operation.

Recovery was uneventful, and the antral balloon was removed on July 22, 1955. To facilitate this procedure and to prevent excessive pressure on the bony walls of the antrum for too long a period, 2 c.c. of saline solution was withdrawn from the balloon every other day,

beginning on July 12, 1955. Postoperative roentgenograms (Fig. 4) and manual palpation revealed good apposition of the fragments. The patient was discharged from the hospital on July 23, 1955 (Fig. 5).

Results.—Postoperative results were excellent. The normal contours of the orbit and zygomatic area were maintained as closely as possible. No loss of vision occurred. Healing of the fractured left ankle was also uneventful.

On Jan. 12, 1956, when the patient last visited the dental clinic, roentgenograms showed good healing of the fracture sites (Figs. 6 and 7).

Fig. 7.—Roentgenogram taken six months after operation shows leveling of the orbital floor and good healing of the fracture sites.

COMMENT

Because of the positioning of fragments in such cases as the one just described, it is frequently impossible to use the towel-clip technique for elevation of depressed zygomaticomaxillary and facial complex segments. Headcaps are cumbersome, and external pin fixation may result in unsightly dermal scarring. If the antrum is in any way involved by the fracture, it should be cleansed thoroughly and old blood clots and bony debris should be removed to ensure more complete recovery. Since this cleansing of the antrum is necessary in many cases, it is relatively simple to introduce an antral balloon at the same time to elevate the depressed segments into a more nearly normal position. The

patient suffers minimal discomfort, and the fracture sites heal promptly without undue difficulties.

This technique, as presented, is simple and uncomplicated. It is an ideal adjunct in the treatment of zygomaticofacial compound dislocations. Even in more involved situations that call for extreme measures in the reduction and fixation of the fractured parts, this procedure offers an excellent means of supporting and maintaining the normal bony contours.[13]

SUMMARY

1. A case has been presented to illustrate the use of the Shea-Anthony antral balloon to reduce a fracture of the zygomaticomaxillary complex.

2. Because of the antral involvement that is present in a high percentage of these cases, cleansing of the antrum and introduction of the balloon can be done as part of the same operative procedure.

3. Adequate clinical and roentgenologic examinations are essential for the correct diagnosis of zygomaticofacial compound dislocations.

4. It must be remembered that competent neurologic and ophthalmologic examinations prior to surgical interference are imperative in these cases.

5. The minimal discomfort experienced by the patient, the lack of dermal scarring, the need for no cumbersome appliances, and the prompt bony healing of the fracture area make this technique an adjunct to be considered in the treatment of zygomaticofacial compound dislocations.

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11 Asylum St.

ANNOUNCEMENTS

American Academy of Oral Pathology

The Academy announces that its officers for 1961-62 are as follows:

President, David F. Mitchell
President-Elect, Norman H. Rickles
Vice-President, George Greene, Jr.
Secretary-Treasurer, Robert J. Gorlin
Editor, Donald A. Kerr

Annual meetings will be held in Miami, Florida, in 1963 and at the National Institutes of Health in Bethesda, Maryland, in 1964.

American Board of Oral Pathology Announces Certification Examination

The next examination by the American Board of Oral Pathology will be given on Dec. 9 and 10, 1961. Candidates will be notified of the place upon being accepted for the examination. All applications for the December examination must be completed by Aug. 1, 1961. Information and application blanks may be obtained from the secretary, Dr. William G. Shafer, Indiana University School of Dentistry, 1121 West Michigan St., Indianapolis 2, Indiana.

American Academy of Implant Dentures

The third annual International Seminar on Implants in Dentistry will be held at the University of Pennsylvania School of Dentistry in Philadelphia, Pennsylvania, Oct. 13 to 15, 1961.

One day will be devoted to a complete demonstration of the two-stage operation involved in the construction and insertion of an implant. The University's closed-circuit television facilities will be utilized for this purpose.

All members of the dental profession are welcome. There will be a fee of $50.00. Application forms and further information may be obtained from Dr. A. Norman Cranin, Chairman of the American Academy of Implant Dentures, 2120 Ocean Ave., Brooklyn 29, New York.

American Academy of Periodontology

The Academy will hold its forty-seventh annual meeting aboard the *M. S. Bergensfjord,* sailing from Philadelphia to Bermuda on Oct. 20 and returning to Philadelphia on Oct. 25, 1961. In Bermuda the ship will serve as the Academy's hotel.

Any member of the American Dental Association or recognized affiliate organizations may secure space on the *M. S. Bergensfjord* as long as space is available.

Those interested in participating in this meeting should contact Mr. E. M. Baskette, Caribbean Cruise Lines, Suite #301, Roeper Bldg., Miami 32, Florida.

Twelfth Annual Berkshire Conference in Periodontology and Oral Pathology

Major General Joseph L. Bernier, Assistant Surgeon General and Chief of the Dental Corps, United States Army, will be one of the principal speakers at the Conference, which will be conducted at Eastover in Lenox, Massachusetts, June 18 to 22, 1961. Other lecturers on the Conference faculty will be Finn Brudevold, Irving Glickman, Reidar F. Sognnaes, John B. MacDonald, Sigurd Ramfjord, Everett Shapiro, David Weisberger, and Frank M. Wentz.

The Conference is open to general practitioners and dentists in specialized practice. Information may be obtained from Tufts Berkshire Conference, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

American Institute of Oral Biology

The Institute will hold its eighteenth annual meeting at the Hotel Riviera in Palm Springs, California, Nov. 6 to 10, 1961. The following outstanding scientists will participate:

W. Ross Adey, M.D., Professor of Anatomy and Physiology at the University of California at Los Angeles. A neurophysiologist of international reputation, Dr. Adey, is recognized for his work on the rhinencephalon, a part of the brain with important influence on the processes of emotion, learning, and memory.

M. Michael Cohen, D.M.D., Professor of Pediatrics at the School of Dental Medicine and Medicine, Tufts University. He will present his work concerning developmental disorders of the dentition.

Russell D. Coleman, D.D.S., Assistant Professor of Anatomy at the School of Dentistry, University of California, San Francisco. He has done important work in the experimental production of orofacial anomalies in animals.

Thomas K. Cureton, Ph.D., Director of the Physical Fitness Laboratories, University of Illinois. Dr. Cureton was exceptionally popular two years ago with his work on physical fitness and is returning by unanimous request; he will conduct special physical fitness testing clinics in addition to his formal presentations regarding the improvement of psychological states by means of physical exercise.

Francis L. Lederer, M.D., Professor of Otolaryngology and head of the department at the University of Illinois, Chicago. Dr. Lederer is internationally known and has written several books in his field. His discussions will include orofacial syndromes and the significance of speech deviations.

William Lefkowitz, D.D.S., Director of Graduate Studies at the University of Kansas City and very well known to the dental and medical professions. His presentations will deal with tissue culture, the development of human enamel, and the transplantation of cultured and uncultured tooth buds.

Jens J. Pindborg, D.D.S., Dr. Odont., Professor of Oral Pathology at the Royal Dental College, Copenhagen, Denmark, and a scientist with a world-wide reputation in his field. He will discuss the pathologic conditions occurring in the periodontal tissues.

For further information and application forms, please write Marion Lewis Meyer, Executive Secretary, 2240 Channing Way, Berkeley 4, California.

New York Institute of Clinical Oral Pathology, Inc.

The next course in clinical oral pathology will be given on two Wednesday evenings a month from October, 1961, through May, 1962. The sixteen sessions will be held from 7:30 to 10 P.M.

There will be lectures on embryology, hematology, histology, and pathology (illustrated by lantern slides, blackboard demonstration, microscopic slides, etc.), and their clinical interpretation. Case reports stressing history, clinical findings, x-ray studies, diagnosis, treatment (including diagnostic and surgical procedures), histopathologic evidence, and prognosis will be presented by the staff of the Institute and by guest speakers.

The course is approved by the Board of Education of the University of the State of New York.

Inquiries should be addressed to the Executive Secretary, 101 East 79th St., New York 21, New York.

Southern California Academy of Oral Pathology

The Academy is a study group of dentists interested in maintaining and increasing their knowledge and experience in pathology. Monthly programs are held on the third Tuesday at the Rodger Young Auditorium, 936 West Washington Blvd., Los Angeles, California. Reservations can be made by writing or calling Dr. Arthur Barrett, 22030 Sherman Way, Canoga Park, California, DIckens 6-5616. The program consists of a dinner meeting followed by a presentation on a basic science or on a clinical subject related to pathology.

German Society for Maxillofacial Surgery

The eleventh annual meeting will be held in Stuttgart, Germany, July 24 to 26, 1961, under the direction of Prof. Dr. K. Schuchardt. The main subject to be discussed is "The Surgical Treatment of Damaged Oral Tissue by Irradiation." In addition, various papers and motion pictures may be presented.

European Stomatologic Association for Implant Dentures

The Association announces that its third International Congress will be held at the New Faculty of Medicine in Paris, France, July 6 to 8, 1961. Those who desire to participate may write to A. E. O. S. I., 3rd Congrès International, Secrétariat Technique, S. O. C. F. I., 1 Ter, Rue Chanez, Paris 16°, France.

German Index of Dental Literature

An index of dental periodicals of the entire world will soon be finished. The linen-bound 110-page book will list 1,200 dental periodicals, together with the exact title, the year of first appearance, the number of issues published each year, the precise address, and the accepted international abbreviation (ISO) for each. Subscription price prior to delivery is DM 15,— (thereafter, DM 20,—). Editors of every periodical are asked to submit specimens of their publications for photographic registration to Stuttgart-Degerloch, Leonorenstrabe 25.

Balint Orban Memorial Seminar

The Seminar (formerly known as the Rocky Mountain Dental Seminar) will be held in Boulder, Colorado, June 25 to 28, 1961. The theme for the Seminar is "Clinical and Microscopic Pathology of the Periodontium and Other Oral Structures." The outstanding group of essayists includes Drs. S. N. Bhaskar, Henry Goldman, Maynard K. Hine, and Harry Sicher.

Further details and application may be obtained by writing to Mrs. James Manella, Secretary, Balint Orban Memorial Seminar, 1700 Mesa Rd., Colorado Springs, Colorado.

Boston University School of Medicine

The Oral Surgery Section offers a one-year oral surgery course planned to meet the requirements of the American Board of Oral Surgery. It provides a foundation for clinical training, such as clinical internship and residency, leading to qualification for the practice of oral surgery as a specialty. This course, given annually, is designed to give, by lecture, clinical demonstration, operative clinic, and laboratory exercise, a systematic review of the clinical phases of oral surgery. This is coordinated and correlated with the pertinent preclinical sciences. The fundamental principles of diagnosis and treatment of the various manifestations in the field of oral surgery are covered, with specific emphasis on etiology and pathology. Instruction is given in oral surgery, principles of general surgery, animal surgery, radiology, general and oral pathology, oral medicine, physical diagnosis, medicine, dermatology, clinical laboratory procedures, anesthesia, physiology, biochemistry, pharmacology, anatomy (head and neck), bacteriology, surgical prosthesis, and oncology. The course is under the direction of Dr. Kurt H. Thoma, Professor of Oral Surgery.

The Orthodontics Section offers a course of two academic years intended for those who wish to prepare themselves for the practice of orthodontics as a specialty. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of the specialty. Courses are given in the preclinical sciences pertaining to this field. Applications are being accepted for the program which begins in the fall of 1962. The course is under the direction of Dr. Herbert I. Margolis, Professor of Orthodontics.

The Prosthetics Section offers a one- or two-year course in general prosthetic dentistry. It is intended for those who wish to prepare themselves for the practice of prosthodontics as a specialty and/or who may wish to prepare for the specialty board examinations. This course consists of lectures, clinical demonstrations, operative clinics, and laboratory exercises in all clinical phases of the field. These are coordinated and correlated with pertinent preclinical sciences. The course is under the direction of Dr. Leo Talkov, Assistant Professor of Prosthetic Dentistry.

The Endodontics Section offers a one-year course to dentists wishing to specialize in the field of endodontics. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of this specialty. Instruction is given in the preclinical sciences pertaining to this field. The course is under the direction of Dr. Herbert Schilder, Assistant Professor of Endodontics.

The Oral Pathology Section offers a two-year course intended to prepare dentists for teaching or research in this field. The program includes the pertinent basic sciences and clinical oral medicine. The related curriculum consists of anatomy, biochemistry, physiology, microbiology, oncology, dermatology, general pathology, and oral pathology. The general pathology is given in connection with the Pathology Department of the Massachusetts Memorial Hospitals.

For application or information, please apply to Department of Stomatology, Boston University School of Medicine, 80 East Concord St., Boston 18, Massachusetts.

Albert Einstein College of Medicine
Yeshiva University

The Department of Dentistry will sponsor a five-day participation course in clinical periodontics Oct. 30 to Nov. 3, 1961. This course will embrace the theoretical and practical application of the causative factors, diagnosis, prognosis, and surgical management of periodontal disease. The course, which is basically designed for out-of-town practitioners, will be under the direction of Irving Yudkoff and Marvin N. Okun. Tuition is $175.00.

For further information, please write Director, Postgraduate Dental Program, Albert Einstein College of Medicine, Eastchester Road and Morris Park Avenue, Bronx 61, New York.

University of Illinois

The College of Dentistry will offer a postgraduate course entitled "Current Histology for the General Practitioner" on June 26 and 27, 1961, on the near west side Chicago Professional Colleges campus. The course is designed to give the general practitioner a refresher program in histophysiology so that dental terminology and the latest concepts in current literature will be clarified in relation to their application in his daily practice. Tuition for the course is $50.00.

Application forms may be obtained from the Division of Postgraduate Education, University of Illinois College of Dentistry, 808 S. Wood St., Chicago 12, Illinois.

University of Texas Dental Branch

Dr. E. C. Hinds, chairman of the Department of Surgery, has arranged a postgraduate course on "Diagnosis and Treatment of Temporomandibular Joint Disease" for June 22 and 23, 1961. Dr. Welden E. Bell of Dallas, Texas, lecturer in oral diagnosis at Baylor University College of Dentistry, will discuss the etiology, diagnosis, and treatment of temporomandibular joint disease. There will be eight lectures, with time allowed for discussion.

The course is acceptable for 16 hours of credit by the Texas Academy of General Dentistry. The tuition fee will be $75.00, of which $50.00 must accompany the application.

Further information may be obtained from Dr. Sumter Arnim, Director, Postgraduate School of Dentistry, P. O. Box 20068, Houston 25, Texas.

University of Washington

The Graduate Division of the School of Dentistry announces a two-year course in endodontics and microbiology leading to a master's degree in dentistry. The course, which will begin in September, 1961, is under the direction of Dr. John I. Ingle.

The trainee will be supported on an annual stipend of $5,300.00 received from a National Institutes of Health Teacher Training Grant. An extensive research project and thesis are required.

Inquiries should be directed to Dr. Saul Schluger, Director of Graduate Dental Education, University of Washington School of Dentistry, Seattle 5, Washington.

REVIEWS OF THE LITERATURE

ABSTRACTS OF CURRENT LITERATURE

Postoperative Vomiting. J. E. Riding. Proc. Roy. Soc. Med. **53:** 671, December, 1960.

The frequency and severity of postoperative vomiting in different circumstances and the factors that influence it are little understood.

To standardize an investigation of postoperative vomiting, patients were interviewed on the day after operation by specially instructed nurses.

Preoperative medications were studied with respect to combinations. Following the use of 10 mg. morphine and 0.6 mg. atropine, sickness was reported in 35.2 per cent; when atropine alone was used, the incidence was only 11.5 per cent; and postoperative vomiting occurred in 66.7 per cent of the patients who had morphine alone. The proportion of sick patients increased as the dose of morphine was increased, with or without atropine. Increasing the dose of atropine decreased sickness, regardless of whether or not morphine was given.

Perphenazine was found to have a good sedative action with a marked antiemetic effect.

Aberrant Salivary Tumors: A Review With Presentation of Six Cases. W. M. Trible. Laryngoscope **71:** 159, February, 1961.

In this report, six cases of aberrant salivary gland lesions are reported. One tumor found in the nasal cavity, was diagnosed as "malignant mixed tumor"; another from the middle turbinate was originally reported as "mixed tumor, histologically benign" and later as "adenocarcinoma." Case 3 involved an ulcerated mass of the left tongue diagnosed as "adenocarcinoma," and in the other three cases the patients had tumors of the palate. Four of the tumors were malignant; one was benign, but recurrent within three years following initial removal; and one was "quite benign."

The author feels that the histologic classification of salivary tumors has been complicated by terminology and by the difficulty of correlating the microscopic picture with the clinical one. All agree that the treatment of choice is surgical intervention.

Accidental Isolated Primary Vaccinia of the Tongue. E. P. Scott and A. J. Steigman. J. Pediat. **58:** 77, January, 1960.

There is a bit of "Scotland Yard" evidenced in this interesting report. A 5-year-old child was vaccinated. Seven days later a second child bit the first child on the vaccination site and developed a sore on the tongue. The second child took a nursing bottle from a third child, sucked on it, and forced it back into the mouth of the third child who, in turn, developed a lesion of the tongue. The diagnosis of traumatic inoculation of vaccine virus was made.

The authors state: "The present position of her vaccination scar is accessible to inspection but may not be as acceptable to school health inspectors and others as a scar in a more orthodox deltoid location."

Routine Treatment of Tetanus. M. A. Perlstein, M. D. Stein, and H. Elam. J. A. M. A. **173:** 1536, August, 1960.

Treatment has been improved by the use of meprobamate to control spasms and by better care of the respiratory tract.

After the initial sensitivity test, 40,000 units of tetanus antitoxin is given intramuscularly and an additional 40,000 units is given intravenously. Penicillin is the most effective antibiotic and is given intramuscularly in a dose of 600,000 units daily.

One of the most important aspects of the total care of the patient with tetanus is the provision of a patent airway by tracheotomy in the severely ill patient.

Nutrition is maintained by intravenous feeding for the first twenty-four hours and then by high-calorie tube feeding.

Cardiac Arrest. L. H. Stahlgren and J. Angelchik. J. A. M. A. **174:** 226, September, 1960.

At the Philadelphia General Hospital in Philadelphia, Pennsylvania, forty-five patients suffered cardiac arrest in the operating room. Of these, twenty-seven (60 per cent) were resuscitated and eleven (24.5 per cent) recovered. Nine exhibited no evidence of neurologic damage, and one had mild hemiparesis. One patient bled to death following rupture of a pre-existing aneurysm. Four patients survived for periods ranging from one week to four months, all with serious neurologic sequelae.

Of twenty-five patients who suffered arrest outside the operating room, seven (28 per cent) were resuscitated, but only one patient recovered and showed no evidence of neurologic sequelae.

Unless cardiac arrest is witnessed by a physician who makes a prompt diagnosis and immediately opens the patient's chest, more than the vital three or four minutes will usually elapse and serious and irreparable damage will be suffered. When it is known that the safe time interval has been greatly exceeded, it is believed that the patient's chest should not be opened. There is no reason to perform resuscitative measures in a patient who, because of a fatal disease, cannot survive even if his heartbeat is restored.